KASR EL - AINI
INTRODUCTION TO SURGERY

SEVENTH EDITION
NEW EDITION

2011

VOLUME 2

DEDICATION

To those who preceded us and are no longer with us
To those who preceded us and are still among us
And to those who will follow us
We dedicate this book



PREFACE

As is clear from the dedication of this book, the driving feeling behind this effort has been a sense of belonging to this chain-like continuum of generations that our hospital Kasr El-Aini has seen passing within its walls, each generation giving way to a new one, but not before producing an indelible mark of its own, unique to it and forever influencing subsequent generations.

Each year sees a few hundred young men and women treading their first steps in medical learning while at the same time a few hundred others will leave this venerable institution armed with the knowledge they have acquired within its walls and with which they are going to start their journey along the arduous path of medical practice.

To us teachers the most enjoyable and certainly the most fascinating aspect of our profession is the actual moulding of minds and personalities that we help to bring about between the start and the end of medical learning. This process has been likened, and not unjustly, to the moulding of a piece of clay by the hands of a sculptor into a coherent and well balanced piece of statuary. It is not therefore surprising that as soon as the idea of this book was formulated, we all jumped with alacrity at this opportunity of expanding the knowledge and directing the thoughts of our students along the proper path.

In this book each of us has tried in his own field and in his own way to present the subject in question in a way that will help the student acquire easy and readily accessible knowledge and will also help him to grasp the unity of medical knowledge, a thing so vital to a deep understanding of the significance of surgery.

In more practical terms much emphasis has been laid on physiology, anatomy and pathology thus never presenting surgery as an isolated science. The clinical picture of each disease has been discussed at great length and its relation to abnormal physiology and anatomy made quite clear while the details of sophisticated operative procedures have been omitted. Again and adhering to the same philosophy we have discussed accident and emergency surgery at great length seeing that it is these problems that the young surgeon will probably have to face early in his career. All the recent advances in the surgical and para-surgical sciences have been included in the text in a clear and concise manner.

In short we have tried to produce a textbook of surgery that medical students as well as young surgeons will find useful.

We thus present our work hoping that those who preceded us will look upon it with approval and that those who will follow us will carry on after us and dedicate newer versions of this book to us who will have preceded them.



PREFACE TO THE THIRD EDITION

The first two editions of Kasr El-Aini Introduction to Surgery were favourably received by both students and colleagues. Meanwhile, we kept open for their criticism and suggestions. We felt that they are sincere in their desire to develop the book that carries the name of our medical school.

Accordingly it was decided that the theme of this new production would be "a student-friendly edition". The content was both updated and trimmed down to suit what an undergraduate needs to know about surgery. We also focused on making knowledge easily picked up by providing more figures, tables, and reminder boxes. A new page format was introduced to serve this purpose.

In this edition the anaesthesia chapter has been removed. Our colleagues in anaesthesia department have produced their own book "Anesthesia for Medical Students". We strongly recommend this book as a companion to Kasr El-Aini Introduction to Surgery.

We hope that the third edition will be more useful and enjoyable to our students. Their comments and suggestions will always be considered for future refinement of the book.

The editors



PREFACE TO THE FIFTH EDITION

The first editions of this surgery manual were met by enthusiasm from students and from faculty members. We have also received some useful criticism, and plenty of useful ideas and suggestions.

For these reasons we were prompted to produce this fifth edition. Most of the chapters have changed, some of them minimally, while a few were completely revised. Moreover, a new chapter about "minor procedures" was added. In this edition the changes included addition of recent advances, deletion of outdated and redundant knowledge, and trimming of some operative details that are not relevant to the undergraduate curriculum. Above all, the focus was on making this edition more reader-friendly. In this context we hope that it will allow for easy understanding, easy recall of knowledge, and wiser application in a clinical setting.

We believe that the fifth edition of Kasr El-Aini Introduction to Surgery is a useful book for the undergraduate medical student, as well as for the young surgeon in training. Furthermore, it provides the essential knowledge that is needed for surgeons who seek higher qualifications.

We wish to express our gratitude to colleagues from neurosurgery, orthopaedic surgery, urology and anaesthesia who participated generously by writing the corresponding chapters.

We wish to express our great appreciation to the Ciba Geigi Pharmaceutical Company represented by the Ciba Scientific Office, Cairo, Egypt for providing us with plenty of very illustrative figures which are of considerable value. All the X-ray pictures were provided by the surgical staff.

We are deeply indebted to all the staff of Al-Ahram press who did their best to bring out this book in an elegant form.

We are very thankful to Mr. Sayed Mahmoud, the director of the University Book Center, for publishing this book.

Finally we would like to thank our students for their enthusiasm and encouragement.



PREFACE TO THE SEVENTH EDITION

The undergraduate medical student of today is in a difficult situation. This is because the evolution of surgical science has become too fast to follow. Furthermore, the practicing surgeon is required to adopt a practice that is based on the best available medical evidence, a duty that requires exhaustive research, sometimes in situations that require a fast precise decision making.

We, at the department of general surgery of Cairo University still believe that a regularly- updated good textbook is a great help to student and to practicing surgeon alike. Though a heavy task, we decided to accept the challenge and to update this book. The success of previous editions, the encouragement of our colleagues in the profession and the unlimited support we got from our chairmen prompted us to do it.

In this edition old knowledge has been removed and all chapters were updated according to the principles of “evidence-based medicine. Some chapters have been expanded to encompass the tremendous advances made in interventional radiology, oncology, laparoscopic surgery, bariatric surgery and molecular biology.

In addition, and in order to prepare our students and junior surgeons to meet the realities of modern practice, we have added two new chapters; pre and postoperative management of the surgical patients and principles of modern surgical practice.

We would sincerely like to thank our students for their useful comments and suggestions and thank all contributors for generously sharing their knowledge and experience.

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THE LIVER

CHAPTER CONTENTS

Surgical anatomy

The largest solid organ in the body is the liver. It weighs 1200- 1800g fl adults.

Position

The liver is wedge shaped and fills the right hypochondrium, and the epigastric region. It extends into the right lumbar region and occupies part of the left hypochondrium,

Description and relations

The liver has two surfaces, parietal, and visceral that are separated inferiorly by a sharp lower border.

Fig. 31.1. Parietal surface of the liver.

Parietal surface (Fig. 31.1)

- The parietal surface is related, in its most part, to the diaphragm, and to a lesser extent to the anterior abdominal wall.
- The diaphragm separates this surface from the pleural cavities, lungs, heart, and lower right ribs.
- The inferior vena cava (IVC) indents its posterior aspect.
- The parietal surface is covered by peritoneum, except for a small bare area to the right of the vena cava sulcus, and at the attachments of the peritoneal ligaments.
- The falciform ligament is a peritoneal fold that connects the parietal surface of the liver to the diaphragm and anterior abdominal wall. It contains the round ligament (ligamentum teres) in its lower free border.
- High up at the dome of the liver, the falciform ligament is continuous to the left with the left triangular ligament, and to the right with the coronary ligament and then the right triangular ligament.

Fig. 31.2. Visceral surface of the liver and its relations.

Visceral surface (Fig. 31.2)

- The left part of the visceral surface is related to the abdominal oesophagus, and anterior surface of the stomach.
- Further to the right, the gall bladder is adherent to the under surface of the liver in what is called the gall bladder fossa.
- The part of the liver between the round ligament and this fossa is called the quadrate lobe.
- The quadrate lobe and the gall bladder are related to the first and second parts of the duodenum, and to the right (hepatic) flexure of the colon.

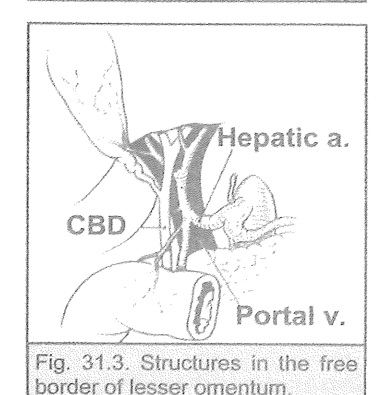


Fig. 31.3. Structures in the free border of lesser omentum.

- The right part of the visceral surface bears the renal impression which is caused by the right kidney and suprarenal gland. The peritoneal space between the right kidney and the right lobe of the liver is called the hepatorenal

(or Morisons) pouch, and is the most dependent part in the peritoneal cavity in the recumbent position.

Lesser omentum

- The lesser omentum is formed of a fold of peritoneum that has a free right border, and connects the lesser curvature of the stomach with the visceral surface of the liver.
- The free border of the lesser omentum contains three important tubular structures (Fig. 31.3)
  - o The common bile duct in an anterior plane and to the right.
  - o The hepatic artery in the same anterior plane but to the left of the duct.
  - o The portal vein posterior to the other two structures,
- As they approach the liver, these three tubes divide into right and left branches each and then enter the hilum of the liver which is known as the porta hepatis.
- The caudate lobe of the liver is just behind the porta hepatis.
- The aditus to the lesser sac is bounded anteriorly by the free border of the lesser omentum, superiorly by the caudate lobe, posteriorly by the IVC, and interiorly by the first part of the duodenum.

Fig. 31.4.Hepatic veins

Fig. 31.5. Plane of division into two equal surgical lobes.

Blood supply and venous drainage

- **Hepatic artery from coeliac trunk.**
- **Portal vein** is formed by union of superior mesenteric and splenic veins behind the neck of pancreas.
- **Three hepatic veins** (Fig. 31.4) that drain into IVC.
  - o Right hepatic vein ends directly in IVC.
  - o Left and middle hepatic veins unite to form a short trunk before joining IVC.

Lymphatic drainage

The lymphatics of the liver drain into lymph nodes at the porta hepatis and eventually to the coeliac lymph nodes. Some lymphatic vessels pierce the diaphragm and empty in the thoracic duct in the thorax.

Segmental anatomy

Grossly the liver seems to be divided into a large right lobe and a small left lobe by the falciforn, ligament on the parietal surface, and by the ligamentum teres and venosum on its visceral surface.

Plane of division

From the surgical point of view, the liver is divided into two almost equal lobes by the middle hepatic vein, the plane of division lies to the right of the falciforn ligament and passes from the gall bladder fossa to the inferior vena cava (Fig. 31.5).

Separate blood supply, venous drainage and bile ducts of surgical lobes

- Each of the two lobes of the liver is supplied by a separate branch of the hepatic artery and the portal vein, and its bile is drained by a separate bile duct.

- There are three hepatic veins. The middle one drains blood from both sides, while the right and left veins drain the corresponding liver lobes.

It thus seems that, the body has two livers that are grossly united, but can be surgically separated and any of them excised without interference with the other.

Sectors and segments (Fig. 31.6)

- The hepatic veins and their tributaries divide the lobe into sectors, and the sectors into smaller segments. Each of these segments has its own smaller branch of bile duct, hepatic artery, and portal vein.
- There are four sectors divided into eight segments, and any of them can be excised if its duct and vessels are ligated and divided.
- The liver segments are numbered from one to eight. Segment one is the caudate lobe that has a special status as it is supplied by hepatic artery and portal vein branches from both sides, and is drained directly by multiple small veins into the inferior vena cava,

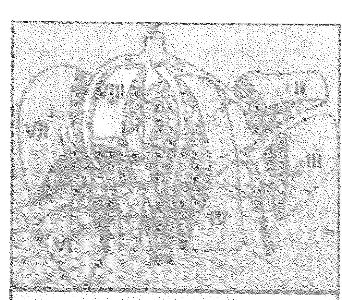


Fig. 31.6. Liver segments. Segment I is the caudate lobe. It does not show on this figure as it lies posteriorly.

Microscopic anatomy

- The liver is covered by the Glisson's capsule, which sends a sleeve extension around the structures in the porta hepatis as they pass inside the liver. This sleeve of connective tissue continues to surround the branches of these structures, even the smallest ones, and forms the portal tracts which appear on histological sections between the hepatic lobules (Fig. 31.7).
- Each portal tract contains a small branch of the hepatic artery, portal vein, and hepatic duct.
- The hepatic lobules are formed of sheets of hepatocytes separated by sinusoids, with a central tributary of a hepatic vein.
- Blood passes from the portal tract in the periphery to the sinusoids and then to the central vein. Reticuloendothelial cells of Kupffer are scattered along the sinusoids.
- Bile is secreted by the hepatocytes and is collected by small canaliculi that deliver it to the branch of bile duct that lies in the portal tract.

Fig. 31.7. Liver histology.

Surgical physiology

The liver is a very busy organ, and it, therefore, receives 1.5 L of blood per minute. The portal vein supplies two thirds while the hepatic artery supplies one third of this amount. Because of its higher oxygen content, the hepatic artery supplies 50% of the oxygen requirements.

Functions

1. **Formation and secretion of bile.** Bile salts, cholesterol, and phospholipids are formed in the hepatocytes. The hepatocytes also pick up unconjugated bilirubin from the blood stream and conjugate it to glucuronic acid to form conjugated bilirubin, which is another constituent of bile. To the above mentioned constituents, bicarbonate and water are added to complete the task of bile formation. Bile reaches the intestine by way of the common bile duct that opens in the duodenum. In the intestine it emulsifies fat prior to its digestion, allows absorption of fats, vitamin K and other fat soluble vitamins, regulates intestinal motility, and provides

the natural brown colour of stools. Bile salts, in addition, have mild antiseptic properties and prevent the intestinal bacteria from being absorbed into the portal circulation. Lack of bile salts in the intestine in cases of obstructive jaundice is known to cause vitamin K deficiency with consequent hypoprothrombinaemia and haemorrhagic tendency. Its lack, also is accompanied by septicaemia, shock, and renal failure which are the common causes of death in cases of obstructive jaundice. Most of the bile salts in the intestine are absorbed in the terminal ileum, reaching the liver to be re-excreted in bile; thus affecting an enterohepatic circulation.

2. The liver is the site of **carbohydrate, protein, and** fat metabolism. Glycogen is formed, and stored in the liver, and is then converted to glucose and released to the circulation in case of need. Proteins as albumin, prothrombin, and fibrinogen are formed. It is also the place where glucose is formed from proteins by the process of gluconeogenesis, and urea is formed by deamination of amino acids. A great part of energy is generated in the liver from the Kreb's cycle, part of which is stored as adenosine triphosphate (ATP).
3. Metabolism of many drugs and hormones, e.g. oestrogens.
4. Removal of ammonia from the portal blood.
5. The liver is the storage house of glycogen, vitamin B_{12}, vitamin A, iron, and copper.
6. Reticuloendothelial cells clear the blood from the bacteria that can escape from the intestine to the portal circulation.

Regenerative hyperplasia

- The liver cells have a remarkable capacity for regeneration by hyperplasia when a part of it is damaged by disease or is surgically resected.
- This ability is greatly diminished in cirrhotic livers. Surgical resection of a part of a cirrhotic liver carries a bad prognosis because of the diminished capacity of regeneration, and because of the poor function of the remaining part.

Studies of the liver

(A) Liver function tests (LFTs) Table (31.1)

Table (31.1) Liver function tests

Test	Normal range
Tests which assess the synthetic functions of the liver	
- Serum albumin	35-50 gm/L
- Prothrombin time (PT)	10-15 seconds
- Partial thromboplastin time (PTT)	35-40 seconds
Tests which assess the excretory functions of the liver	
- Alkaline phosphatase	30-110 IU/L
- 5'-Nucleotidase	
- Gamma-glutamyl transferase	10-50 IU/L
Tests which denote liver cell injury	
- Serum aspirate aminotransferase (AST)	5-40 IU/L
- Serum alanine aminotransferase (ALT)	5-40 IU/L
Serum bilirubin total and direct is a test of the hepatocellular and excretory functions	
• Total bilirubin	0.3-1.0 mg/100 ml
• Direct bilirubin	0.35 mg/100 ml

N.B. Alkaline phosphatase may be raised in bony or intestinal problems.

(B) Imaging of the liver

1. Ultrasonography is the first choice imaging investigation of the liver because it is simple, non-invasive, reliable, not expensive, and is easily repeated. It is particularly useful for detecting focal lesions, and allows for guided biopsy.
2. Computed tomography (CT scan) and magnetic resonance imaging (MRI) visualize the parenchyma and adjacent tissues with great clarity.
3. Arteriography is now loosing popularity in favour of the previously mentioned non-invasive techniques. It is still, however, useful in the diagnosis of liver tumours and to display the vascular anatomy before liver resections.
4. Isotope scanning of the liver was previously in common use to detect space occupying lesions of the liver. It has been nearly replaced by ultrasonography or CT scan.

(C) Liver biopsy

Indications

1. Target biopsy of focal lesions that are suspected to be primary or secondary tumours is sometimes indicated. Haemangiomas and hydatid cysts should not be biopsied.
2. Liver transplant assessment. A preoperative histological diagnosis is essential, and the technique can also help in the early detection of postoperative transplant rejection.

Techniques

1. The standard method is the percutaneous technique. To reduce bleeding, the prothrombin time should not be prolonged more than 3 seconds above the control value, and the platelet count should be above 80000/uL. Even so, it is safer to cross match two units of blood to be ready in case of bleeding. The procedure is done under local anaesthesia, usually through the right midaxillary line just below the upper limit of liver dullness. The commonly used needles are the aspiration (Menghini) and the cutting (Tru-cut) types. To biopsy a focal lesion, passage of the needle is guided by ultrasound or CT scan.
2. Laparoscopic biopsy (Fig. 31.8).
3. Liver biopsy at laparotomy. In diffuse liver disease it is customary to obtain a deep wedge biopsy from the lower border of the right and the left lobes. An intraoperative needle biopsy from the depth of liver substance further increases the diagnostic accuracy.

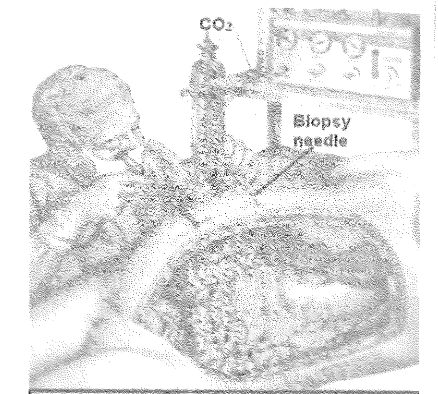


Fig. 31.8. Laparoscopic liver biopsy.

Liver trauma

- The liver is the second common solid abdominal organ to be injured. It is preceded only by the spleen.
- Liver injuries are commonly associated with affection of other intra or extra-abdominal organs. The ribs, pleura, lungs, colon and spleen are common associations.
- The prognosis after treatment of liver injury largely depends on these associated injuries.

Aetiology

1. **Accidental trauma that is either blunt as** in road traffic accidents (RTA), or penetrating as with stabs and bullets. The damage inflicted by the latter is directly proportionate to its velocity, so the most harmful are the high velocity missiles of rifles as they shatter the liver substance.
2. **Iatrogenic injury** is increasing with the rising popularity of invasive investigations as percutaneous liver biopsy, and percutaneous transhepatic cholangiography (PTC).
3. Spontaneous rupture of the liver is an extreme rarity that may happen with eclampsia or hepatic tumours.



Fig. (31.9) Deep tear of right lobe of live

Pathology

Type of injury

In increasing seriousness the following types can be seen

1. Small subcapsular haematoma.
2. Small superficial tear or tears.
3. Large subcapsular or intrahepatic haematoma.
4. Large deep tear or tears (Fig. 31.9)
5. Shattered liver parenchyma which may include a whole lobe (Fig. 31.10).
6. Vascular injury, the most difficult to control is that of the main hepatic veins because of the difficult access.



Fig. 31.10. Shattered right lobe of liver. This is a postmortem specimen. The patient died of severe haemorrhage.

Consequences

1. The main danger of such injuries is bleeding, and this should be the main concern of the surgeon. Most liver injuries stop bleeding by the time they are explored, but some of them cause death from blood loss.
2. A liver haematoma sometimes communicates with a torn bile duct allowing blood to trickle down the biliary passages to gastrointestinal tract producing what is known 'haematobilia',

Remember

- **Liver injury can be associated with affection of other organs, inside and outside the abdomen.**
- **Prognosis of liver injury depends largely on the presence of associated injuries.**
- **Priorities of trauma management (Chapter 2).**
- **Chest stabs as high as the 4th intercostal space can transfix the diaphragm and injure the liver (and / or the spleen).**

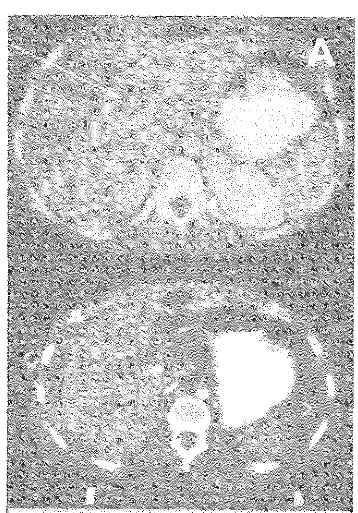
Clinical features and diagnosis

Liver injuries draw attention to their presence because of intraperitoneal haemorrhage.

1. History of trauma.
2. Abdominal pain.
3. Abdominal tenderness and rigidity are caused by parietal peritoneal irritation, either by blood or escaping gastrointestinal secretions.
4. The presence of lower rib fractures raises the suspicion of liver affection.
5. Massive bleeding presents with the picture of haemorrhagic shock.
6. Minor bleeding is discovered by diagnostic peritoneal lavage (DPL), ultrasound or by CT scan (Fig. 31.11), which are done in suspected cases. These tests are particularly useful in the unconscious patient as it is difficult to assess the abdomen.
7. The injury may also be discovered with systematic exploration during laparotomy for penetrating abdominal trauma.

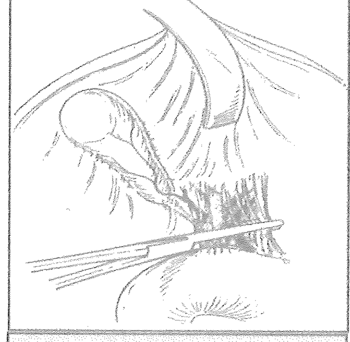
Treatment

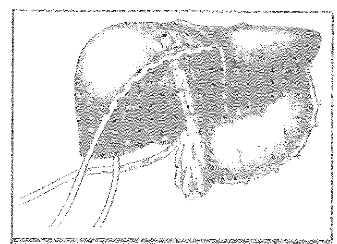
1. The priorities of multiple trauma management, that are mentioned in chapter 2, should be followed.
2. Patients who are haemodynamically stable with no evidence of peritonitis, are treated conservatively by repeated examination, CT scan may be repeatedly performed.
3. Patients who are haemodynamically unstable, have peritonitis or deteriorate under conservative treatment will need laparotomy.


Fig. 31.11. CT scans that show liver injuries.
A. Extensive laceration of Rt. Lobe.
B. Rt. lobe tear with haemoperitoneum (arrows).

Principles of surgical management

1. Adequate exposure by a longitudinal incision that can be extended to the chest in case of need.
2. Thorough systematic exploration of the abdomen is required to assess the liver affection and to detect other intra-abdominal injuries.
3. Priority is for arrest of bleeding. As mentioned before, most small liver tears are found to have stopped bleeding by the time the abdomen is explored, and these tears deserve no treatment Preliminary control of brisk liver haemorrhage can be attained by a combination of temporarily packing the bleeding area, and the application of Pringle's manoeuvre (Fig. 31. 12). The latter entails application of a vascular clamp to the free border of the lesser omentum, or holding it between two fingers, to occlude the hepatic artery and the portal vein for a period up to 20 minutes. The lessened rate of bleeding allows the surgeon to visualize and ligate the injured vessels.
4. Whenever possible suturing liver tears should be avoided because it is likely to leave a space for accumulation of haematoma that may infect or communicate with intrahepatic bile ducts. It is, however, resorted to if control of bleeding vessels is not possible in deep tears. Tying sutures over pedicled omentum helps haemostasis (Fig. 31.13). Deep transverse mattress sutures using special liver needles is recommended.
5. A haematoma is explored to ligate the damaged vessels and ducts, and to excise the dead tissues. It is then left open for drainage.
6. A lobe that is shattered beyond salvage is treated by excision of this lobe.
7. Firm packing of inaccessible and difficult bleeding areas, e.g., the hepatic veins, may be the only method for temporary arrest of bleeding (Fig. 31.14). The patient is transferred to a specialized centre where the pack is removed in the operating theatre, and the injury is dealt with.
8. Multiple intraperitoneal drains are always placed to guard against collections of blood and bile (Fig. 31.13). Prophylactic antibiotics are prescribed.


Fig. 31.12. Pringle's manoeuvre.


Fig. 31.13. Deep sutures over pedicled omentum. Drains are always inserted.

> **Important points to remember regarding liver injuries**
> - **Suspect liver injuries in patients with fractures of the right lower ribs.**
> - **Minor liver injuries can be treated conservatively.**
> - **Pringle's manouever is useful for temporary control of hepatic bleeding during the operation.**
> - **Perithepatic packing is very useful as a last resort to stop bleeding in serious hepatic injuries.**

The mortality of liver injury averages 15-20%. It gets worse if other major organs are injured.

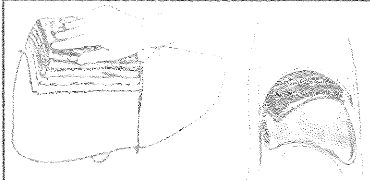


Fig. 31.14. Packing for inaccessible bleeders.

Infections of the liver

The following are the liver infections with surgical interest

1. Viral hepatitis (see chapter 7).
2. Pyogenic liver abscess.
3. Amoebic hepatitis and abscess.
4. Hydatid disease of the liver.
5. Hepatic Schistosomiasis.

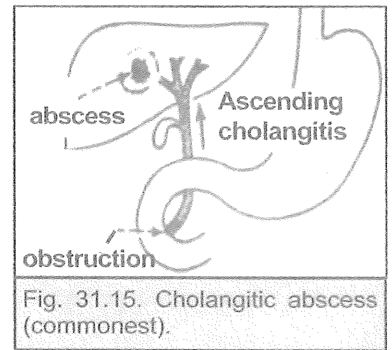


Fig. 31.15. Cholangitic abscess (commonest).

Pyogenic liver abscess

This is a serious highly fatal disease. The key to improving the results of its treatment is an early diagnosis. The development of modern diagnostic and interventional radiology, together with the evolution of antibiotics, contributed to a safer management of the problem.

Aetiology

(A) Sources of infection

a. **Biliary tract (cholangitic abscess)**. This is the commonest of all sources. Ascending cholangitis caused by bile duct obstruction results in multiple abscesses of the liver (Fig. 31.15). The likely organism is E. coli or other gram negative bacilli.

Fig. 31.16. Portal pyaemia with multiple liver abscesses.

b. **Portal vein**. Suppurative appendicitis or colon diverticulitis may induce septic thrombophlebitis of the portal vein radicles, with consequent portal pyaemia and multiple liver abscesses (Fig. *31.16).* Streptococci and anaerobes are the common organisms. Today, fewer patients present with portal pyaemia, probably because of early diagnosis of intraperitoneal sepsis and the widespread use of potent antibiotics.

c. **Arterial system (haematogenous abscess)**. The source may be bacterial endocarditis, tonsillitis, intravenous drug misuse, or osteomyelitis. Staphylococcus aureus is the usual causative organism.

d. **Idiopathic**. The source of infection is not possible to trace.

(B) Predisposing factors

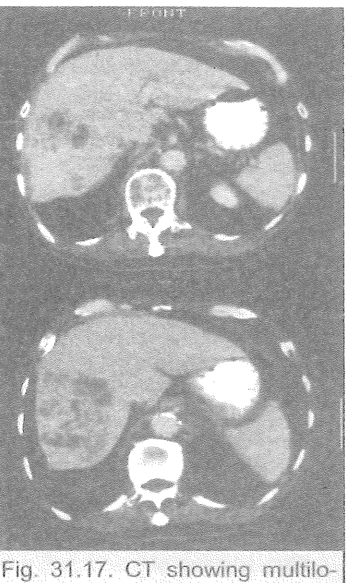
a. The immunocompromized patient, e.g., diabetes mellitus, leukaemia, chronic illness, the elderly, the alcoholics, and transplanted patients receiving immunosuppressive therapy.
b. An already existing liver lesion as hydatid cyst, amoebic abscess, or haematoma rarely gets secondarily infected.

Complications

Direct extension of the abscess to neighbouring tissues may involve the pleura, lung, pericardium, or peritoneum.

Clinical features

- The usual symptoms include fever and its constitutional manifestations, toxaemia, together with right hypochondrial or lower chest pain.
- Abdominal examination usually reveals a tender hepatomegaly.
- This may, however, be absent and the patient just presents with pyrexia of unknown origin (PUO).
- Sometimes the features of the causative lesion are evident, e.g. the Charcot's triad of cholangitis, or the pain of acute appendicitis.


Fig. 31.17. CT showing multilocular liver abscess.

Investigations

1. The usual laboratory disturbances include
   a. Leucocytosis.
   b. Anaemia and high ESR.
   c. Low serum albumin.
   d. High alkaline phosphatase and transaminases level.
   e. Elevated serum bilirubin in cases of cholangitis, or with multiple abscesses.
2. Imaging. Ultrasound or CT scan (Fig. 31.17) can accurately diagnose and localize the abscess.

Treatment

Broad spectrum antibiotics are prescribed
"As for any abscess the treatment is drainage of pus".

- There is now a trend to depend on percutaneous guided drainage of pus. The procedure is a simple one that is done under local anaesthesia, where ultrasound or CT is used to direct the needle towards the abscess cavity. The abscess is aspirated and then a tube drain is inserted.
- This procedure has nearly replaced the standard method of open surgical drainage.

Amoebic hepatitis and abscess

This disease is commoner than the pyogenic abscesses in developing countries, particularly those lying in the tropical and subtropical regions. The low standards of hygiene, coupled with high humidity favour amoebiasis infestation.

Aetiology

Amoebic hepatitis and abscess are due to infection with the protozoal parasite, Entamoeba histolytica. The condition is a complication of amoebic colitis.

Pathology

Gross picture

- The trophozoites invade portal blood from the colon, and migrate up to reside in the liver (Fig. 31.18).
- The right lobe of the liver is affected more than the left. This is explained by the observation that portal blood from the right side of the colon drains mainly to the right lobe of the liver while that from the left colon drains to the left lobe. As amoebic colitis affects mainly the right colon, it is expected that amoebic hepatitis affects mainly the right side.
- The commonest site of amoebic abscess is the posterosuperior segment of the right liver lobe. Again this is explained by the segmental divisions of the portal vein as the segmental branch of the right portal vein to this segment is in direct continuity with the right portal vein.
- The parasite starts a process of liquefactive necrosis, and when there is heavy infection the necrosis results in the formation of an abscess that is usually single and unilocular.
- Pus is usually sterile with a brown chocolate colour, and is sometime pinkish, in which case it is likened to 'anchovy sauce". The abscess has a shaggy wall that harbours the amoeba.

Amoebic abscess
Portal drainage
Amoebic colitis

Fig. 31.18. Origin and spread of amoebic liver abscess.

Complications

- Secondary bacterial infection.
- Rupture into the pleura, lung, pericardium, or peritoneum. When the abscess ruptures upwards, it usually leads to a lung abscess rather than empyema because the two layers of the pleura are usually obliterated.

Clinical features

Symptoms

- Upper abdominal pain.
- Low-grade fever. This is in contrast with the high-grade fever that is seen with a pyogenic abscess.
- Anorexia, nausea and weight loss.
- A clear history of an attack of dysentery is not always obtained.

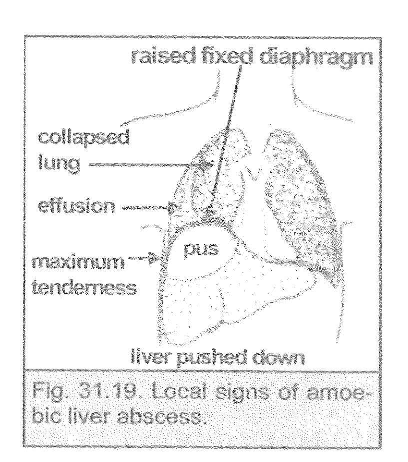


Fig. 31.19. Local signs of amoebic liver abscess.

Signs (Fig. *31.19)*

1. The patient is usually pale and looks toxic, a combination that gives him an earthy look.
2. Tender hepatomegaly.
3. There may be tenderness in the lower right intercostal spaces.
4. Chest-examination may reveal right basal lung abscess or right pleural effusion.

Differential diagnosis

1. Pyogenic liver abscess.

2. Hepatocellular carcinoma in black Africans assumes a rapidly progressive course with pain and constitutional symptoms that simulate those of an amoebic abscess.

Investigations

1. Positive stool analysis and serological tests are useful diagnostic aids in non-endemic areas. In countries where amoebic colitis is prevalent, these tests are likely to be positive in a high percentage of the population.
2. Blood picture. There is commonly leucocytosis and anaemia
3. Similar to pyogenic abscess, the diagnosis rests mainly on imaging by ultrasound or CT scanning which can detect the number, site and size of the abscess.
4. A chest X-ray is needed to detect pleural effusion and pulmonary collapse. It also commonly reveals an elevated right cupola of the diaphragm (Fig. 31.20).
5. The high success of metronidazole treatment is made use of as a therapeutic test. An improvement in the general and local conditions after three days of treatment confirms the diagnosis.

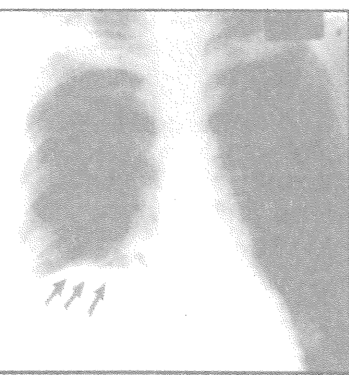


Fig. 31.20. Elevated right hemidiaphragm and pleural effusion are seen on plain chest X-ray in a case with amoebic liver abscess.

Treatment

1. Conservative treatment is highly successful. Metronidazole is the drug of choice. It is given in a dose of 800 mg three times daily for 7-10 days.
2. Ultrasound-guided percutaneous aspiration is needed for abscesses that fail to respond to treatment within 72 hours, and for large abscesses. Aspiration is done by a large bore - spinal needle under local anaesthesia. The site of aspiration depends on the site of the abscess; if anterior the needle is introduced below the costal margin anteriorly, and if posterior it is inserted in the 10^{th} intercostal space posteriorly. Few days later ultrasound is repeated and if the abscess cavity has recollected and is more than 5 cm, aspiration is repeated.
3. **Open drainage**

   Indications
   - Presence of secondary infection.
   - If the abscess is pointing.
   - If aspiration is difficult because of multilocular abscess or the presence of thick pus.

   As most of amoebic abscesses are in the postero-superior segment of the right lobe, open drainage which is rarely needed, is done through the bed of the 12^{th} rib posteriorly through an extrapleural approach.

Hydatid disease of the liver

Geographical distribution

Hydatid cysts of the liver are prevalent in sheep-rearing parts of the world. In the Middle East the disease is commonly seen in Iraq, Yemen, and Libya; but is sometime also met within Egypt (Chapter 7).

Aetiology

Causative agent

The disease is caused by the tapeworm Echinococcus granulosus.

Life cycle (Fig. 31.21)

- The adult worm lives in the intestine of the dog, which is the definitive host.
- Ova are passed in the dog's stools, and may be ingested by sheep as they feed on contaminated grass.
- Once in the sheep's stomach, the ova hatch, penetrate the wall and enter the portal venous system to lodge and form cysts mostly in the liver, and less frequently in the lungs or brain. Sheep are thus considered to be the secondary host, Man may, by accident, become a secondary host through vegetables or hands that become contaminated with dog's excreta.
- The parasite's life cycle is continued when dogs feed on the offal of dead sheep.

Definitive host
Main site
Ova expelled
Intermediate host

Fig. 31.21. Hydatid life cycle.

Pathology

Gross picture

- The liver may be the site of a single or multiple hydatid cysts.
- The cyst fluid is usually colourless and clear, but may be yellowish if the cyst establishes a communication with the bile ducts.
- Scolices are found in this fluid, and, therefore, may cause a severe anaphylactic reaction if it gets to the circulation.

Microscopic picture (Fig. 31.22)

- The parasite forms a double-layer cyst wall. The inner thin layer (endocyst) is formed by an inner germinal layer and the outer laminated membrane. The endocyst shows folds forming brood capsules that contain the heads of future worms (scolices). This is the living part of the parasite that secretes the hydatid fluid.
- The host reacts to the intrusion of the parasite by surrounding the cyst by a third outermost adventitial fibrous tissue layer.
- Daughter cysts may form inside the main cyst.

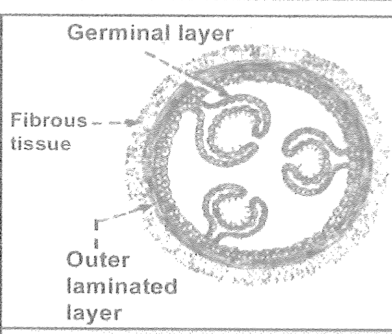


Fig. 31.22. Three layers of cyst.
- Germinal and laminated layers belong to parasite.
- Fibrous tissue layer belongs to host.

Fate and complications

1. The fate of these cysts is quite variable.
    - The majority of cysts continue to enlarge gradually.
    - In others the parasite dies and the cyst is calcified (Fig 31.23).
2. A small proportion is affected by complications that endanger the patient's life.
    - Secondary infection
    - Rupture of the cyst in the biliary passages causing jaundice
    - Rupture in the peritoneal cavity causing dissemination and an anaphylactic reaction.

Clinical features

1. The cysts may be asymptomatic for years and may be discovered with ultrasonography that is done for another purpose.
2. The usual presentation, however, is by chronic right upper quadrant pain and hepatomegaly.
3. Rarely the disease presents by one of its complications.

4. In 70% of cases, the cyst is felt as a well-defined painless fluctuant swelling, which often exhibits a hydatid thrill on palpation due to vibration of daughter cysts.

Investigations

1. Blood picture may show is eosinophilia.
2. Ultrasound or CT scan (Fig. 31.24) shows the well-circumscribed cyst (or cysts). In an endemic area, a liver cyst is usually caused by hydatid disease. Daughter cysts may be also visualized,
3. The intradermal Casoni's test is now superseded by the more sensitive haemagglutination, (ELISA) test or complement fixation test which detects hydatid antibodies in the serum. The disadvantage of Casoni's test is that once positive, it remains so for the rest of the patient's life. It also gives false positive results in 40% of cases.

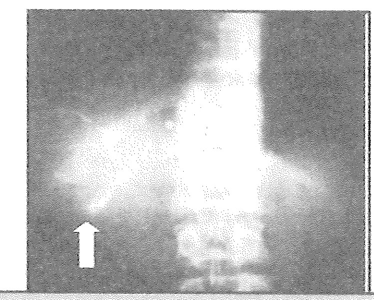


Fig. 31.23. Calcified hydatid.

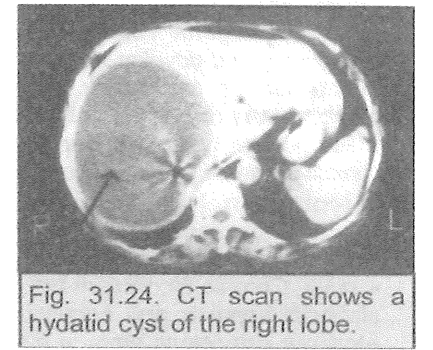


Fig. 31.24. CT scan shows a hydatid cyst of the right lobe.

Treatment

1. Except for small asymptomatic cysts, the treatment is by surgical removal.
    a. Accurate preoperative estimate of the number and sites of the cysts is needed.
    b. The danger with hydatid cyst excision lies in the possibility of hydatid fluid spillage into the peritoneal cavity. This mishap carries the double risk of peritoneal implantation of the scolices forming other cysts, and of absorption into the circulation inducing a life threatening anaphylactic reaction.
    c. When the liver cyst (or cysts) is approached, it is surrounded by dark green towels that are moistened with a scolicidal agent such as hypertonic sodium chloride or povidone iodine (betadine). The dark green colour makes a suitable background that allows visualization of any spilled scolices.
    d. The cyst is first aspirated and hypertonic sodium chloride is injected to make it three quarters full.
    e. The overlying liver substance and the surrounding adventitia are incised, and the double layered cyst is easily enucleated.
    f. Small cysts need no further treatment, but the cavity left by a large cyst is filled with pedicled omentum.

    Some authors now advocate a policy of percutaneous aspiration and injection of hypertonic saline or alcohol.

2. **Medical treatment:** Treatment with mebendazole or albendazole is not a good substitute for surgery, but is used in the following indications.
    a. Treatment of the elderly frail patients.
    b. Treatment of recurrent cases.
    c. Some authorities advise its routine postoperative use to prevent recurrence. The usual dose of mebendazole is 400-600 mg three times daily for one month.

Liver cirrhosis

This is a condition in which the whole liver is replaced by multiple nodules separated from one another by anastomosing sheets of fibrous tissue. Cirrhosis is the end result of the following pathological processes

1. Long continued loss and necrosis of liver cells.
2. Persistent inflammatory reaction.
3. Regeneration of the liver lobules in an irregular manner with loss of architecture leading to the formation of large number of nodules. The portal tracts and hepatic veins are spaced irregularly in the nodules.
4. Extensive fibrosis.
5. Fibrosis and architectural distortion interfere with blood flow through the liver leading to continuous loss of liver cells even in the absence of the original insult which caused the cirrhosis.

To summarize cirrhosis entails necrosis, regeneration and fibrosis.

It was previously thought to be an irreversible disease, it now appears that with treatment of the underlying insult, (e.g. chronic hepatitis C, alcohol abstinence, haemochromatosis) there can be some reversal of fibrosis.

Aetiology of liver cirrhosis

1. **Post-viral**. This is now the most common cause of cirrhosis in Egypt and it can occur in association with bilharzial periportal fibrosis. Some patients (about 6%) who get viral B or C hepatitis do not recover completely and develop chronic active hepatitis. There is piece-meal necrosis of the liver, where there is destruction of the liver cells at the interface between connective tissue and parenchymal cells. Lymphocytes or plasma cells infiltrate between liver cells. The end result will be cirrhosis usually of the macronodular type. This type of cirrhosis usually occurs at a younger age than alcoholic cirrhosis and its clinical progress is rapid. There is also a higher incidence of liver cell carcinoma.
2. **Alcoholism**. High and continuous alcohol consumption is a common cause of cirrhosis especially in western countries. It is estimated that a daily dose of 100 gm of alcohol represents the hepatotoxic level. Alcohol can produce cirrhosis on its own without any nutritional deficiency but the latter will aggravate its toxicity. Chronic alcoholism will lead to fatty liver. About 1/3 of patients with fatty liver will progress to alcoholic hepatitis and about 1/3 of patients with alcoholic hepatitis will be complicated by cirrhosis. Alcoholic cirrhosis is more in males usually between 40-70 years of age. The cirrhosis is usually of the micronodular type in the early stages but later it may show the mixed or the macronodular varieties. Alcoholic cirrhosis progresses more slowly than most other types and abstinence of alcohol intake may lead to improvement.
3. **Biliary cirrhosis**. This is secondary to long-continued cholestasis due to the harmful effect of retained bile upon the hepatocytes. There are two types:
    a. Primary biliary cirrhosis is of unknown Aetiology: There is lymphocytic and plasma cell infiltration of the small intra-hepatic bile ducts. Later, there is fibrosis of the portal tracts and cholestasis. Eventually irregular loss of liver cells and nodular regeneration complete the picture of cirrhosis. This disease is supposed to be due to an immune reaction, It can be diagnosed by the detection of anti-mitochondrial antibodies and by liver biopsy.
    b. Secondary biliary cirrhosis is due to prolonged mechanical obstruction of the larger biliary passages, e.g. in congenital biliary atresia, sclerosing cholangitis or post-operative biliary stricture. Biliary obstruction will lead to accumulation of bile in the hepatocytes, bile canaliculi and kupffer cells. A

progressive inflammatory reaction develops in the portal tracts followed by fibrous septa linking up adjacent portal tracts. Liver cell necrosis and regenerating nodules develop leading to micronodular cirrhosis.

4. **Congenital cirrhosis**. This is secondary to a group of metabolic abnormalities and include:
   a. **Wilson's disease (Hepatolenticular degeneration)**. This is an autosomal recessive disease in which increasing amounts of copper accumulate in and damage the liver, the kidneys, the lenticular nuclei, and the eyes. The end result will be liver cirrhosis, Kayser Fleischer rings in the cornea and muscular tremors and spasticity.
   b. **Haemochromatosis**. There are excess deposits of iron in hepatocytes, Kupffer cells and portal tract macrophages. Increased amounts of lipofuscin are also present in hepatocytes. The excess iron deposits stimulate excess fibrous tissue formation and eventually cirrhosis. Excess iron deposits occur also in the pancreas leading to diabetes.
   c. $Alpha_{I}$-antitrypsin deficiency and galactosemia.
5. **Nutritional**. Protein deficiency can lead to cirrhosis. The liver in these patients is susceptible to some toxic agents which do not affect the normal liver.
6. **Cryptogenic cirrhosis**. There is no definite cause.
7. **Cardiac cirrhosis** is due to
   a. Long-standing right-sided heart failure.
   b. Budd-Chiari syndrome.
   c. Veno-occlusive disease.

Bilharzial periportal fibrosis

This is not a true cirrhosis. A large number of ova laid in the terminal radicles of the mesenteric vessels fail to engage in the walls of the vessels and form emboli in the portal blood. The ova reach the intrahepatic radicles of the portal vein and escape in the portal tracts where they produce bilharzial granulomas followed by fibrosis. The end result will be excess fibrosis of the portal tracts, a picture simulating cirrhosis. As the criteria of cirrhosis are not present, bilharzial fibrosis is not considered as true cirrhosis.

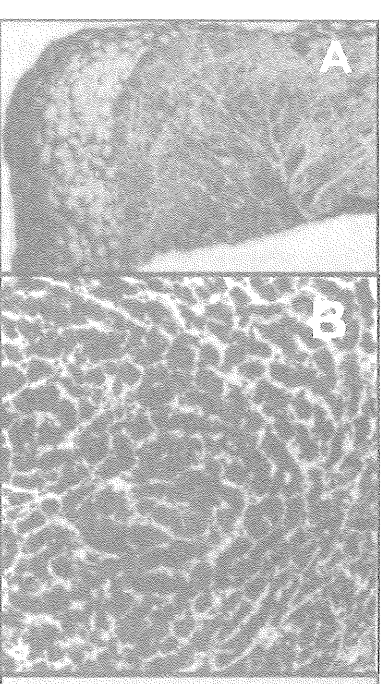


Fig. 31.25. Liver cirrhosis.
A. Gross appearance.
B. Microscopic appearance. Notice the loss of architecture.

Pathology

Gross appearance of cirrhosis

In the early stages the liver may be of a normal size, or enlarged if there is fatty infiltration of liver cells and hyperplastic regenerating nodules. As the disease progresses the liver shrinks due to loss of liver cells and extensive fibrosis.

The surface is nodular and the cut surface reveals that the whole liver is replaced by rounded nodules separated by fibrous tissue (Fig. 31.25A). Cirrhosis is classified according to the gross appearance into

- Micronodular type; the nodules are of the same size and about 3 mm in diameter.
- Macronodular type; the nodules are of variable size and up to 1 cm in diameter.
- Mixed type.

Microscopic appearance of cirrhosis

- The nodules show loss of the normal architecture (Fig. 31.25B). The portal tracts and hepatic veins have lost their regular spacing.
- There are foci of liver cell atrophy and foci of hyperplasia and hypertrophy so that the cells are enlarged and include binucleate forms.
- There are extensive fibrous tissue septa between the nodules composed of fine or dense collagen fibres. The portal tracts and hepatic veins are included in this fibrous tissue.
- Lymphocytes and plasma cells infiltrate the connective tissue and less commonly the parenchymal nodules.

Gross and microscopic appearances of bilharzial periportal fibrosis (Fig. 31.26)

This is classified into 2 types depending on the type of portal tracts involved

(a) **Fine bilharzial periportal fibrosis**. Few ova are deposited in the small portal tracts. The external surface of the liver is nodular; the nodules measure up to 15 mm in diameter and are separated from each other by depressions in which the capsule is opaque and thickened by fibrous tissue. Microscopic examination reveals strands of fibrous tissue enclosing partially or completely one or more of the liver lobules. Dark brown schistosomal pigment may be seen in the kupffer cells. Few bilharzial ova may be seen amidst the fibrous tissue.

(b) **Coarse bilharzial periportal fibrosis**. Large number of ova are deposited in the large portal tracts. Gross fibrous thickening of the affected tracts occurs. The liver is often reduced in size. The surface of the liver presents multiple shallow depressions separating slightly raised areas. The cut surface shows the characteristic thickened large portal tracts (pipe-stem cirrhosis). Microscopy may show bilharzial worms in the portal veins.

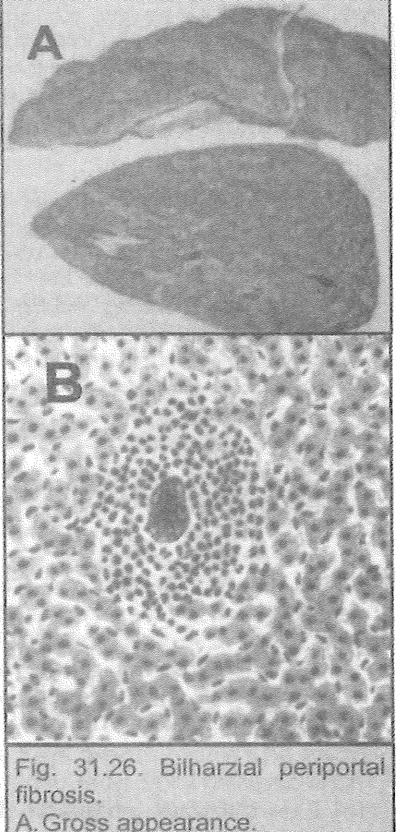


Fig. 31.26. Bilharzial periportal fibrosis.
A. Gross appearance.
B. Microscopic appearance.

It is to be noticed that there is no gross damage to the liver lobules in bilharzial fibrosis. Only some pressure atrophy of the liver lobules may occur in the neigh-bourhood of the thick portal tracts. The essential sequel of bilharzial periportal fibrosis is obstruction of the portal vein radicals in the portal tracts (Presinusoidal obstruction).

Clinical sequelae of liver cirrhosis

Whatever the aetiology of cirrhosis, the patient will suffer from two major problems

(a) Hepatocellular insufficiency due to diminution of functional capacity of the liver.
(b) Portal hypertension-due to interference with the portal blood flow.

Hepatocellular insufficiency

Cirrhosis may remain silent for sometime and is discovered only after routine liver function tests or abdominal ultrasound. In some patients the functional reserve of the liver is adequate to make them look fine, but an insult to the liver due to gastrointestinal

haemorrhage, the use of narcotic drugs, bacterial infection or anaesthesia, can lead to severe parenchymatous damage.

Cirrhotic patients will usually elicit one or more of the following

1. **Anorexia** with loss of weight and ill health. The patient does not have the normal vigour and strength.
2. **Spider naevi**. These are located in the face, neck and upper arms, each consists of a central arteriole from which radiate small vessels.
3. **Palmer erythema**. The hands are warm, the thenar and hypothenar eminences are bright red.
4. **Testicular atrophy** and gynaecomastia occur in males due to failure of the liver to inactivate oestrogens. In females there may be menstrual irregularities, breast atrophy or secondary amenorrhoea, the mechanism of these abnormalities is not known.
5. **Bleeding tendency** leads to epistaxis, bleeding gums, and liability to haematoma formation. There is disturbance in the synthesis of all coagulation factors except factor VIII in addition to thrombocytopenia due to portal hypertension or hypersplenism.
6. **Hyperdynamic circulation** with warm hands and increased cardiac output is probably due to vasoactive substances and due to hypervolaemia.
7. **Jaundice** mild or moderate jaundice is usually present in late cases of cirrhosis. It is a bad prognostic sign.
8. Ascites. It aetiology in cirrhotic patients is multifactorial
   a. Hypoalbuminaemia as the liver is the sole site of albumin synthesis. When the level of blood albumin drops below 3 gm/100 ml, ascites usually develops.
   b. Salt and water retention due to high levels of aldosterone, oestrogens and antidiuretic hormone.
   c. Portal hypertension alone cannot cause ascites.
   d. Increased lymphatic transuclation from the surface of the liver due to lymphatic obstruction.
9. **Hepatic encephalopathy**. This represents a combination of neurologic symptoms and signs accompanying advanced decompensated liver disease and/or extensive portal-systemic shunting. The exact aetiology of this problem is not known, but it is supposed to be due to high levels of ammonia, false neurotransmitter as octapamine, mercaptans and other amines in the blood and cerebrospinal fluid. Disturbance in the ratio between branched chain to aromatic amino acids is also a possible aetiological factor. The patient develops apathy, lack of awareness, lethargy, disorientation and there may be coarse flapping tremors and muscular rigidity. The condition may progress to deep somnolence and finally coma.
10. **Common associations**. Cirrhotic patients have certain clinical association with some pathological
    problems including
    a. Chronic duodenal ulcer.
    b. Chronic pancreatitis,
    c. Enlargement of the parotid glands.
    d. Dupuytren's contracture.
    e. Primary liver carcinoma. About 10% of cirrhotics will develop hepatocellular carcinoma (HCC).

Treatment

Liver cirrhosis

Unfortunately cirrhosis is an irreversible pathology.

- Cirrhotic patients should stick to complete abstinence from alcohol.
- They should avoid intake of any drugs or anaesthetics that are hepatotoxic.
- High carbohydrate intake.
- Vitamins.

Treatment of oesophageal varices is discussed under portal hypertension.

Portal hypertension

The normal portal pressure equals 7 mmHg (8-12 cm of water). The average blood flow to the liver is about 1500 ml/minute of which two thirds come from the portal vein and one third from the hepatic artery. Portal hypertension is present when the portal vein pressure exceeds 20 mmHg (25-30 cm of water).

Aetiology

Portal hypertension occurs mainly due to increased resistance to portal venous flow. The causes (Fig. 31.27) may be classified as:

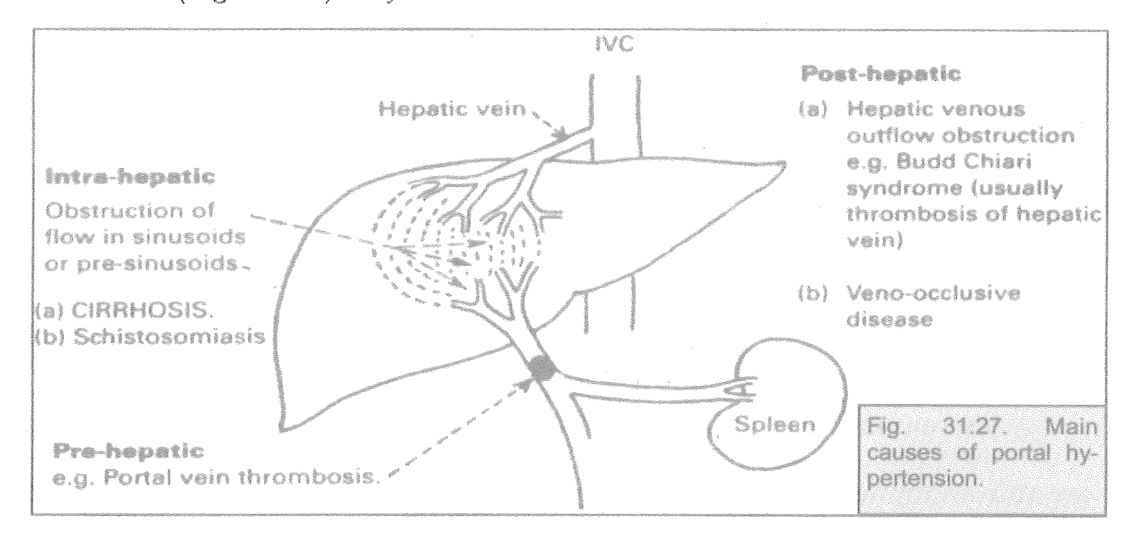


Fig. 31.27. Main causes of portal hypertension.

Pre-hepatic causes

1. Congenital malformation of the portal vein where it is replaced by a cavernomatous mass.
2. Neonatal umbilical sepsis leading to thrombophlebitis of the umbilical vein with consequent obliteration of the portal vein.
3. Portal vein thrombosis secondary to intra-abdominal sepsis, and the use of oral contraceptives or rarely in association with liver cirrhosis, hepatocellular or pancreatic carcinoma, or chronic pancreatitis.

In most of patients with pre-hepatic portal hypertension the liver will be normal.

Intrahepatic causes

1. Liver cirrhosis. Obstruction is sinusoidal and postsinusoidal.
2. Bilharzial periportal fibrosis. Obstruction is presinusoidal.

The causes of portal hypertension in cirrhotic patients are:

(a) Diminution of the total vascular bed by obliteration, distortion and compression of sinusoids.
(b) Compression of the tiny radicles of portal and hepatic veins by the excessive fibrosis.
(c) Development of multiple arteriovenous shunts between the branches of the hepatic artery and those of the portal vein.

Post-hepatic obstruction

1. Budd-Chiari syndrome which is caused by
    a. Thrombosis of the hepatic veins which may be spontaneous or secondary to polycythaemia or the use of oral contraceptives.
    b. Malignant invasion of the hepatic veins.
2. Veno-occlusive disease in which there is obstruction of the hepatic venules.

Sequelae and clinical picture of portal hypertension (Fig. 31.28)

1. **Porto-systemic collaterals**. There are multiple sites of porto-systemic anastomoses, but they are collapsed under normal conditions due to the lower pressure of the portal circulation. With progressive portal hypertension, these collaterals become engorged in an attempt to divert the blood away from the portal circulation. The important sites of these collaterals are
    a. The lower part of the oesophagus and fundus of the stomach between the tributaries of the left gastric and short gastric veins (portal) and the oesophageal veins which drain into the azygos and hemiazygos veins (systemic). These venous plexuses are present in the subepithelial, submucous and perioesophageal layers and their enlargement is called oesophageal or gastric varices. Erosion of the mucosa overlying these varices can lead to bleeding which may take the form of haematemesis, melaena or even fresh bleeding per rectum if the bleeding is massive. The causes which precipitate this bleeding are not exactly known. Of the supposed factors are the high portal pressure, trauma of food or reflux of gastric juice leading to oesophagitis. Bleeding oesophageal varices is the commonest cause of haematemesis in Egypt especially in patients with bilharzial hepatic fibrosis or cirrhosis. It should be stressed that a patient with oesophageal varices may bleed from another lesion, e.g. gastric erosions or peptic ulcer.
    b. In the anterior abdominal wall between the para umbilical vein (portal), if patent and the veins of the anterior abdominal wall as the superior and inferior epigastric veins (systemic). Enlargement of these collaterals can be seen clinically as caput medusae and sometimes auscultation may detect a venous hum due to the increased blood flow.
    c. The lower rectum and anal canal between the tributaries of the superior haemorrhoidal vein (portal) and the middle and inferior haemorrhoidal veins (systemic). This sometimes causes huge congestion of the veins of the lower rectum and the anal canal leading to what is called anorectal varices. If haemorrhoids occur in a patient with portal hypertension they are usually primary.
    d. The retroperitoneurn. Extensive collaterals develop between the tributaries of the superior and inferior mesenteric veins and the veins of the posterior abdominal wall and subdiaphragmatic veins.
2. **Splenomegaly.** Splenic enlargement in patients with bilharzial hepatic fibrosis is at first due to reticuloendothelial hyperplasia due to absorption of bilharzial toxins, Later, with progressive portal hypertension, the spleen gradually enlarges due to congestion and in some cases it reaches the right iliac fossa. Venous congestion of the spleen may predispose to recurrent attacks of splenic infarction causing severe stitching pain. These infarctions heal by fibrous tissue which causes adhesions between the spleen and the diaphragm and the abdominal wall. Both splenomegaly and portal hypertension may lead to secondary hypersplenism.
3. Congestion of **the whole gastrointestinal tract** leads to anorexia, dyspepsia, indigestion and malabsorption. These are the causes of the abdominal discomfort

which is usually present in these patients, and which is wrongly attributed to splenomegaly.

4. **Ascites** the aetiology of ascites is multifactorial as mentioned before. Portal hypertension alone cannot cause ascites. It only localizes the filteration of fluid into the peritoneal cavity.

   Ascites may cause an umbilical hernia. Infection of ascitic fluid may lead to primary bacterial peritonitis.

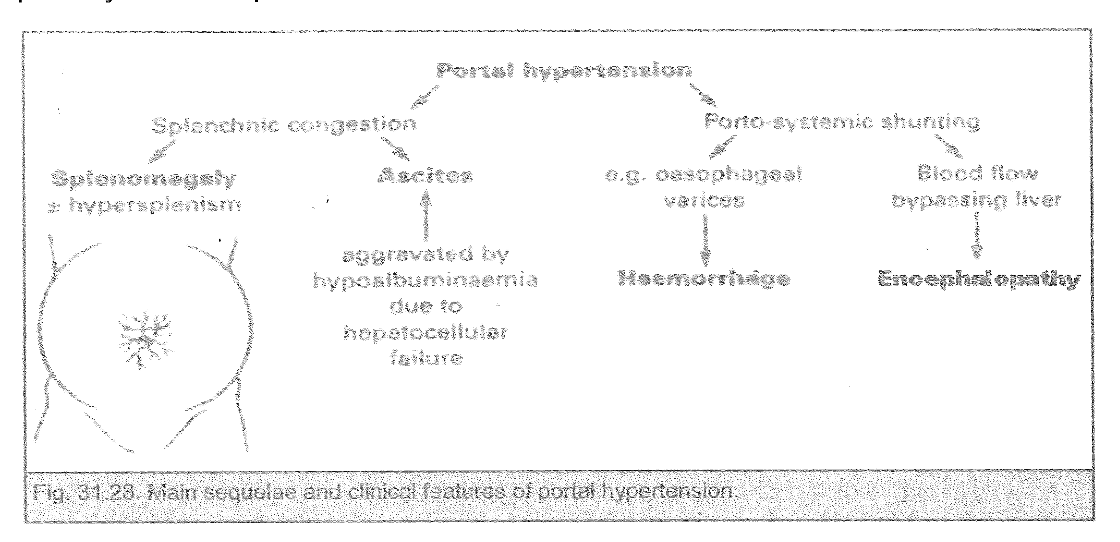


Fig. 31.28. Main sequelae and clinical features of portal hypertension.

Investigations

The aims of investigations in a cirrhotic patient are

1. **Assessment of the functional capacity of the liver is** performed by liver function tests which will reveal
   a. Hypoalbuminaemia. The liver is the only site of albumin synthesis.
   b. ALT and AST are moderately raised.
   c. Prothrombin time and concentration are disturbed. This test is the most sensitive liver function.
2. Detection of **oesophageal varices** can be by either
   a. Fibreoptic upper endoscopy (Fig. 31.29).
   b. Barium swallow can visualize varices in 90% of cases. They appear as multiple, smooth, rounded filling defects (Fig. 31.30).
   c. Duplex scan can show dilated portal vein and collaterals.
3. **Detection of splenic sequestration and hypersplenism**
   a. Blood picture will reveal anaemia, leucopenia, thrombocytopenia or pancytopenia.
   b. Bone marrow examination will reveal hypercellularity.
   c. Radioactive isotope studies using the patients own RBCs tagged with ^{51}Cr will reveal diminished half life of RBCs and increased radioactivity over the spleen. The spleen/liver radioactivity index is increased.
4. **Diagnosis of the aetiology** of liver disease is performed by

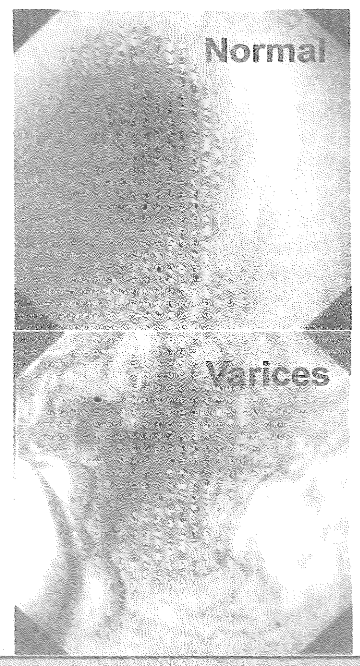


Fig. 31.29. The appearance of oesophagus on endoscopy.

a. Immunological tests for hepatitis markers.
b. Liver biopsy after assessment of prothrombin time and concentration.

To have an idea about the parenchymatous functions of the liver the parameters of Child Pugh classification can provide very useful data (Table 31.2).

Patients with Child A classification can tolerate major surgery. Class C are not candidates for major surgery.

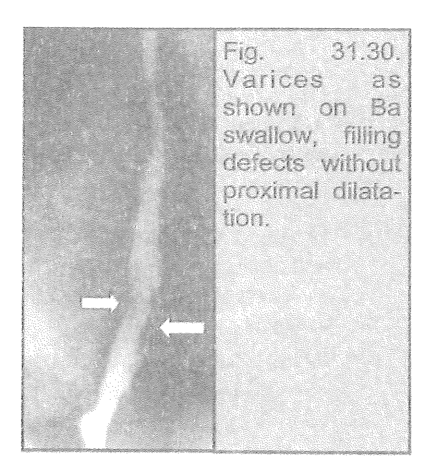


Fig. 31.30. Varices as shown on Ba swallow, filling defects without proximal dilatation.

Table (31.2) Child Pugh classification

Child class	A	B	C
Serum bilirubin mg%	Below 2	2-3	Above 3
Serum albumin gm/100 ml	Above 3.5	3-3.5	Below 3
Ascites	None	Easily controlled	Poorly controlled
Nutritional status	Excellent	Good	Poor
Encephalopathy	None	Minimal	Severe

Treatment

Patients with portal hypertension will belong to one of three categories:

1. Portal hypertension with silent varices.
2. Actively bleeding oesophageal varices.
3. Patients with history of bleeding oesophageal varices.

Silent varices

Prevention of bleeding from silent varices is important since the lifetime risk of a first-time variceal bleed in a cirrhotic patient is 30% and carries a mortality of up to 50%. Endoscopic screening for varices should be done in all cirrhotics.

Primary prevention with a non-selective beta-blocker (propranolol or nadolol) is started if large varices are found, especially if they have stigmata of bleeding (red streaks on the variceal surface). Primary prevention decreases the risk of a first variceal bleed by up to 40%.

Management of patients with actively bleeding oesophageal varices

1. **Admission**. The patient should be admitted to hospital. Even minor haematemesis may be followed by massive bleeding.
2. **Resuscitation**. A wide bore cannula is inserted. A blood sample is taken for complete blood picture, liver function tests, coagulation studies and for cross-matching for at least 4 units of blood. Restoration of the patient's blood volume should be rapid. Overexpansion of the circulation should be avoided as it may lead to heart failure, ascites or renewed bleeding. Morphine and Pethidine are contraindicated in these patients.
3. Correct **coagulopathy**. Vitamin K is administered intravenously. Fresh frozen plasma may be needed.
4. Prevent **encephalopathy**. Blood in the intestine will be fermented to ammonia and other nitrogenous products. These products will be absorbed but they by-pass the

liver through the portasystemic collaterals. Even those which reach the liver are not completely detoxified due to disturbed liver function. The end result is a high level of these nitrogenous products in the systemic circulation which may lead to hepatic encephalopathy. Every attempt should be made to evacuate blood from the gastrointestinal tract and to prevent its fermentation by:

a. Repeated enemas or colonic lavage.
b. Oral lactulose. This is a disaccharide sugar which is fermented by the intestinal flora to lactic acid. The latter combines with ammonia. It also has a mild laxative effect that helps clear blood from the bowel.
c. Neomycin 0.5 gm every 4 hours can reduce the bacterial flora.

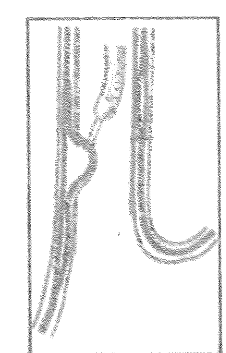


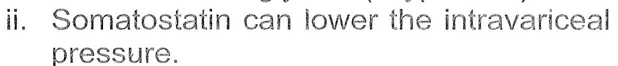


Fig. 31.31. Endoscopic sclerotherapy and band ligation.

5. **Stop bleeding**. Measures to stop the bleeding are arranged in order of priority. If one method fails, the next is used
   a. **Urgent upper GI endoscopy** to confirm the source of bleeding and for Endoscopic sclerotherapy or band ligation (Fig. 31.31). Once the patient is resuscitated, he is taken to the endoscopy department and an oesophago-gastric endoscopy is performed under diazepam. If the varices are not bleeding at the time of examination, the endoscopist should look for other sources, e.g., peptic ulcers. If varices are bleeding, they are injected. There are two methods for injecting varices. In the intravariceal method 5 mls of ethanolamine oleate are injected into each varix just above the oesophagogastric junction to produce thrombosis, For gastric varices intravariceal injection of histoacryl is performed. This substance solidifies after contact with blood. In the perivariceal method, small quantities of aethoxysclerol (0.5 ml) are injected at multiple sites alongside each varix to produce perivascular fibrosis. Both methods may be combined. Injection of bleeding varices is more difficult than in elective cases as the oesophagus may be frill of blood but the result is very gratifying as bleeding stops in 90% of cases. Injection must be repeated at 2 weekly sessions until the varices are obliterated. Side effects of injection sclerotherapy include
      i. Retrosternal discomfort for few days.
      ii. Fever.
      iii. Repeated injections can cause an oesophageal ulcer, which itself may cause bleeding.
      iv. Repeated sclerotherapy may lead to fibrosis and stricture.
      v. Rarely penetration of the oesophagus and mediastinitis.

   Band ligation. The idea is to encircle each varix by a tight band which leads to thrombosis of the veins. It is as effective as sclerotherapy in the control of bleeding, but the number of sessions is less than sclerotherapy. This method is now the first choice for control of bleeding varices.
   b. **Drugs**

i. Vasopressin produces vasoconstriction of the arterioles of the splanchnic circulation and so reduces the portal pressure. Vasopressin is given in a dose of 0.2 unit/kg wt, dissolved in 200 ml of 5% dextrose and given over 20 minutes. Disadvantages of vasopressin are; colicky abdominal pains, diarrhoea and anginal pains, so it is contraindicated in the elderly. Vasopressin can produce temporary control of bleeding. In an attempt to prolong its action it is combined with glycine (Glypressin).



ii. Somatostatin can lower the intravariceal pressure.



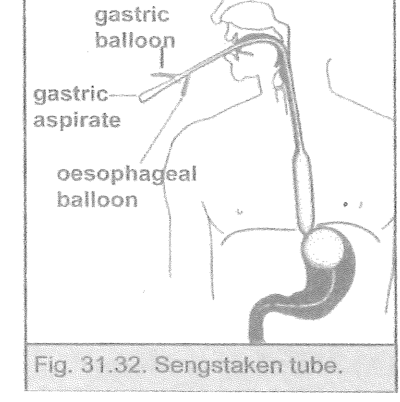


Fig. 31.32. Sengstaken tube.

c. **Balloon tamponade** if bleeding is too profuse to permit endoscopic treatment, balloon tamponade may be used as a temporary measure while arranging for repeat endoscopy or emergency TIPSS or surgery. Sengestaken-Blackmore or Linton tube may be used. The gastric balloon is inflated first by 200 ml of air, and pulled upwards to press the gastric fundus. If bleeding continues, the oesophageal balloon is inflated. The pressure in the oesophageal balloon should not exceed 40 mm Hg. This therapy is effective in controlling bleeding in 80-90% of cases. Disadvantages of balloon tamponade include

i. Discomfort to the patient.
ii. The patient cannot swallow his saliva, continuous aspiration of saliva is important. There are tubes which contain a special channel for aspiration.
iii. Liability to cause oesophageal ulceration or stricture,
iv. Once the tube is deflated, there is liability to rebleeding in 60-80% of patients.

d. **Transjugular intrahepatic porto-systemic shunt (TIPSS)** is the recommended procedures if all the previous measures fail to stop the bleeding. Under fluoroscopic guidance a guide wire is passed through the internal jugular vein to a hepatic vein and then through the liver until it reaches a portal vein branch. A stent is passed along the guide wire until it lodges inside the liver creating a shunt between a portal and hepatic vein (Fig. 31.33). It has proved very effective in the control of variceal haemorrhage.

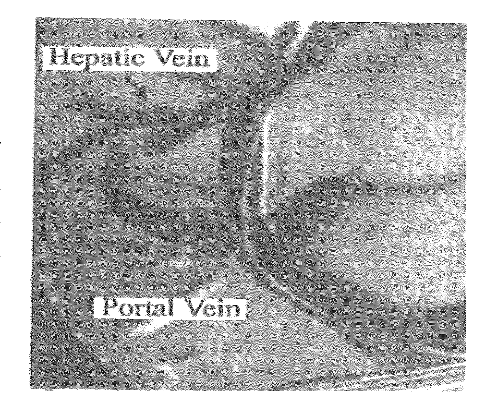


Fig. (31.33) Venogram showing TIPSS

Complications

- Perforation of the liver capsule leading to fatal haemorrhage.
- Porto-systemic encephalopathy in 40% of patients.
- Shunt occlusion 50% after one year.

The results of shunt operations and esophageal stapling in patients with bleeding varices are not satisfactory and have a high mortality.

Treatment of patients with history of bleeding oesophageal varices

Bleeding from esophageal varices represents the major threat to cirrhotic patients. It is usually massive, and once the patient started bleeding, he is liable to have further attacks.

1. Repeated sclerotherapy or band ligation until the varices are obliterated is usually the first choice.
2. Elective surgery is mainly indicated for patients in whom sclerotherapy or esophageal banding failed to stop recurrent attacks of bleeding provided that they are fit.

Fig. 31.34. Portal vein and tributaries

Operations for portal hypertension

Surgical procedures for portal hypertension include:

(A) Shunt operations

The idea of these operations is to lower the portal pressure by shunting the portal blood away from the liver. So long as the portal pressure remains in the normal range, the varices will collapse and bleeding stops.

Knowledge of portal vein anatomy and its tributaries (Fig. 31.34) helps to understand shunt surgery. These operations may shunt the whole portal blood to the systemic circulation (total shunt) or selectively shunt the portal flow away from the area of the lower oesophagus and upper stomach (selective shunt). Transjugular intrahepatic porto-systemic shunt (TIPSS) is a simple alternative to surgery that is done by interventional radiology.

Fig. 31.35. Total shunt surgery.
1. Porta-caval 2. Proximal spleno-renal 3. Mesocaval

Total shunt operations (Fig. 31.35)

1. **Porta-caval operation:** The distal end of the portal vein is divided and transfixed while the proximal end is anastomosed to the anterior surface of the inferior vena cava. The operation is very efficient in lowering the portal pressure and so no further bleeding occurs from the varices. However, this operation has two serious disadvantages
   a. The operation deprives the liver of portal blood flow, which accelerates the onset of liver failure.
   b. Shunting the portal blood completely away from the liver leads to recurrent hepatic encephalopathy in 30-50% of patients.

2. **Proximal spleno-renal shunt** Splenectomy is performed and the proximal end of the splenic vein (the side towards the portal vein) is anastomosed to the left renal vein. It is indicated if the portal vein is thrombosed or if splenectomy is indicated due to hypersplenism. The incidence of encephalopathy following this operation is less than after portacaval shunt, but it is less effective in preventing further bleeding. If the splenic vein is less than 1 cm, the anastomosis is liable to thrombosis.

Bleeding from varices is related to:

1. Size of varices.
2. Bad liver functions (Child C).
3. Wedged hepatic venous (WHV) pressure > 10 mmHg.

3. **Mesocaval (Drapanas)** shunt comprises insertion of a graft whether a synthetic graft as dacron, or autogenic vein between the superior mesenteric vein and the inferior vena cava. The incidence of thrombosis is high.

Selective shunt (Warren shunt) (Fig. 31.36)

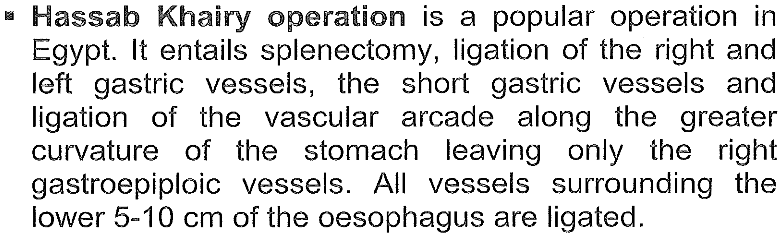
The rationale of this operation is to selectively decompress the critical area of the lower oesophagus without interfering with the portal blood flow. The right and left gastric vessels are ligated. The splenic vein is mobilized, its proximal end is ligated while the distal end is anastomosed to the left renal vein. The short gastric veins are preserved and will selectively decompress the lower end of the oesophagus. The incidence of encephalopathy is low, and the liver functions remain normal.

drainage of varices

Fig. 31.36. Warren shunt.

(B) Porta azygos disconnection operations

The purpose of these operations is to disconnect the collaterals between the portal and systemic circulations which are present in the lower end of the esophagus and the gastric fundus and which are the source of bleeding. There are many techniques for performing devascularization.



- **Hassab Khairy operation** is a popular operation in Egypt. It entails splenectomy, ligation of the right and left gastric vessels, the short gastric vessels and ligation of the vascular arcade along the greater curvature of the stomach leaving only the right gastroepiploic vessels. All vessels surrounding the lower 5-10 cm of the oesophagus are ligated.
    - There is no encephalopathy following this operation and the portal blood flow is intact. There is a low incidence of rebleeding following the operation, but it can usually be controlled by sclerotherapy.

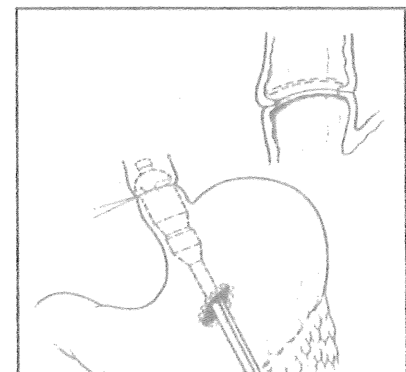


Fig. (31.37) Stapling the lower esophagus

- **Oesophageal transection**. The purpose of this operation is to interrupt the submucosal venous plexus which is still intact following porta azygos dissection. Nowadays, the use of stapling devices has made this procedure simple and easy and it can be added to porta azygos disconnection.

(C) Liver transplantation is indicated for patients with end-stage liver disease. It will correct the problem of the varices and ascites.

Liver tumours

Table 31.3. Classification of liver tumours

Benign	
True neoplasms Non-neoplastic lesions	Liver cell adenoma (LCA) ▪ Haemangioma ▪ Focal nodular hyperplasia
Malignant	
Primary hepatocytes Bile ducts Mixed Secondary	Hepatocellular carcinoma (HCC) Cholangiocarcinoma Cholangiohepatoma From GIT, pancreas or breast cancer.

Liver cell adenoma (LCA)

This lesion is a true benign neoplasm of hepatocytes.

Aetiology

It occurs almost only in women. The use of contraceptive pills is incriminated in its initiation and progress. Cessation of pill intake can result in tumour regression.

Rule of two thirds of portal hypertension

- Two thirds of patients with cirrhosis will develop portal hypertension.
- Two thirds of patients with portal hypertension will develop oesophageal varices.
- Two thirds of patients with oesophageal varices will bleed from the varices.

- All benign liver tumours are commoner in females, and bear variable degrees of causal relationship to the female hormone oestrogen.
- Primary liver cancer is, by far, commoner in men.

Pathology

Grossly LCA is soft, well-circumscribed, and light yellow in colour (Fig. 31.38). It is multiple in one third of cases.

Histologically, LCA is formed of sheets of regular hepatocytes with thin-walled vessels, yet no portal triads.

Complications are rare and include malignant transformation and spontaneous rupture that causes internal haemorrhage.

Clinical features

- The tumour is commonly asymptomatic, and is discovered incidentally at ultrasound examination or at laparotomy.
- Sometimes a large LCA causes right upper quadrant pain.

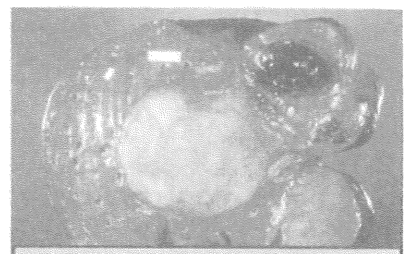


Fig. 31.38. LCA is well circumscribed and is lighter in colour than norma liver tissue.

Investigations

- Imaging by ultrasound, CT scan, and/or MRI,
- Liver function tests and a-fetoprotein are within normal levels.

Treatment

- Liver resection is indicated if doubt exists as to the possibility of malignancy, and for large symptomatic tumours.
- In other cases the contraceptive pill intake is discontinued and the tumour is regularly checked for change of size.

Haemangioma

Haemangiomas are the commonest benign tumours of the liver.

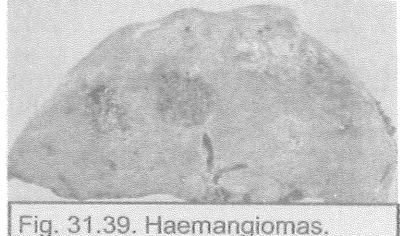


Fig. 31.39. Haemangiomas.

Pathology

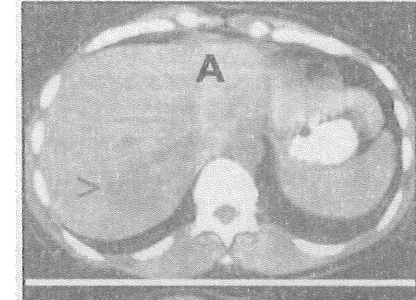
They are of the cavernous type (Fig. 31.39), and may attain a large size. They are usually harmless, yet they occasionally rupture producing serious bleeding.

Diagnosis

Haemangiomas that are discovered as focal lesions on ultrasonography, are correctly diagnosed by either angiography, or CT scan with contrast (Fig. 31.40). Suspicion of a haemangioma is a contraindication for biopsy.

Treatment

Asymptomatic haemangiomas that are diagnosed at laparotomy should not be resected as the natural history is towards involution and calcification. Excision is reserved for the rare lesions that produce pain or bleeding,



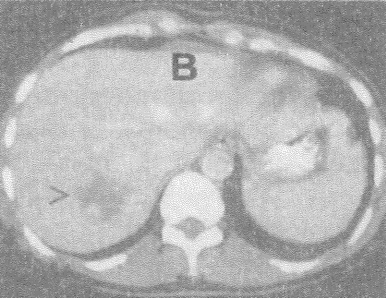


Fig. 31.40. CT scan of liver haemangioma.
A. Without contrast.
B. With IV contrast. The cavernous spaces fill with the contrast material (arrows).

Focal nodular hyperplasia (FNH)

This is a harmless lesion of the liver whose main importance is the difficult differentiation from other focal lesions.

Aetiology

Contraceptive pills stimulate the development and growth of FNH.

Pathology

Macroscopically FNH is a nodular firm mass, which is slightly paler than the normal liver and has prominent surface vasculature. The cut section shows a stellate scar with radiating fibrous septa that divide the lesion into lobules (Fig. 31.41).

Histologically, it resembles cirrhosis with regenerating nodules. The central scar and fibrous septa are apparent.

Diagnosis

The pathognomonic stellate scar can be seen on CT scan. **Sulphur colloid scan** FNH appears as hot area, this differentiates it from hepatocellular carcinoma or metastases.

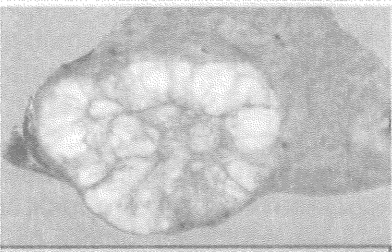


Fig. 31.41. FNH showing the characteristic stellate scar.

Treatment

- Most cases require no treatment.
- Contraceptive pills should be discontinued.
- Cases that are suspicious of malignancy are explored, and subjected to frozen section examination which is diagnostic.

Primary liver cancer

Epidemiology

- In contrast to benign tumours, primary liver malignancies are, by far, commoner in men.
- Hepatocellular carcinoma (HCC) is the main primary liver malignancy and is prevalent in the Far East and in equatorial African nations. The world's highest incidence is in Mozambique.
- Cholangiocarcinoma arising from the intrahepatic bile ducts is much less common than HCC and it also has its highest incidence in the Far East.

Remember
- **90% of HCC are related to viral hepatitis (B or C) or to liver cirrhosis.**
- **Approximately 10% of patients with liver cirrhosis will develop HCC.**

Aetiology

HCC. The following conditions are associated with the development of HCC

1. Infection with hepatitis B virus (HBV), or hepatitis C virus (HCV).

2. **Liver cirrhosis of any cause**. The risk of development of HCC differs with the pathological type of cirrhosis. The highest incidence is with post-hepatitic, followed by alcoholic cirrhosis. These are the two most important aetiological factors as 90% of HCC occurs in patients that are carriers of hepatitis virus or who have cirrhosis. There is evidence that prolonged infection with HBV or HCV results in integration of the viral DNA into the host genome, thus starting the malignant changes.
3. **Aflatoxin ingestion**. This substance is formed by the fungus Aspergillus flavus which grows on grains that are stored in moist warm conditions. The poor storage conditions of grains in Africa favour its growth.

Cholangiocarcinoma: The development of cholangiocarcinoma is related to infestation with the liver fluke Clonorchis sinensis" that cause Asciatic cholangitis.

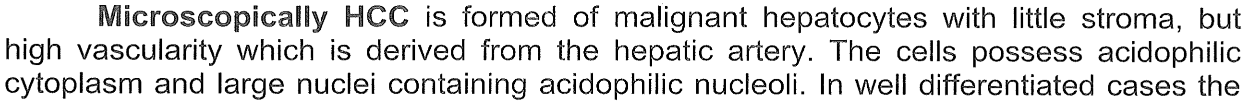
Fig. 31.42. Gross types of HCC.

Pathology

HCC

Gross appearance. The tumour is yellow in colour and commonly arises in a cirrhotic liver. It assumes one of different appearances (Fig. 31.42).

- Massive form, i.e., forming a localized mass. The fibrolamellar HCC is a variety of the massive type that affects non-cirrhotic young females, and hence has a better prognosis.
- Nodular form, i.e., multiple nodules scattered over the liver.
- Diffuse form that is characterized by diffuse infiltration throughout the liver.



Microscopically HCC is formed of malignant hepatocytes with little stroma, but high vascularity which is derived from the hepatic artery. The cells possess acidophilic cytoplasm and large nuclei containing acidophilic nucleoli. In well differentiated cases the cells are strikingly similar to normal liver cells, whereas in anaplastic cases the similarity becomes less striking. The fibrolamellar type is an exception as it contains numerous fibrous septa that make it resemble focal nodular hyperplasia and the cells are deeply eosinophilic.

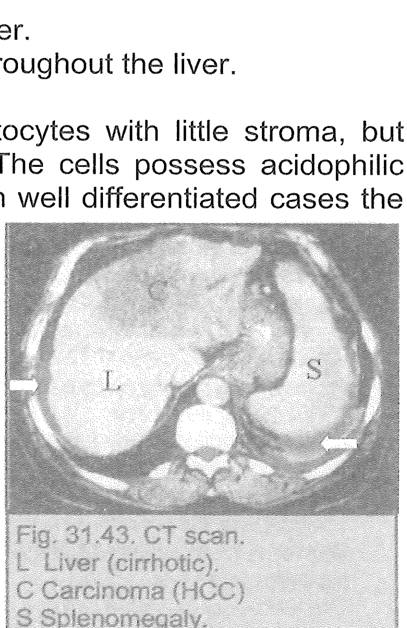


Fig. 31.43. CT scan.
L Liver (cirrhotic).
C Carcinoma (HCC)
S Splenomegaly.
Arrows pont to ascites.

Complications

1. Spread by the lymphatic and venous routes producing porta hepatis nodal enlargement, peritoneal nodules, and less commonly lung deposits.
2. Spontaneous rupture may produce a contained subcapsular haematoma, or massive intraperitoneal haemorrhage.

Cholangiocarcinoma: The tumour is an adenocarcinoma of the lining epithelium of the intrahepatic bile ducts. It spreads widely inside and outside the liver, and by the time the tumour is discovered, the disease is usually so advanced to cure.

Hepatocholangiocarcinoma. This is a mixed type that behaves like HCC.

Hepatoblastoma. This is a form of HCC that occurs in children. It is termed hepatoblastoma because of the similarity to foetal liver cells.

Clinical features

Different types of presentation depend on the *race,* development of the country, and tumour size.

1. Small lesions may be accidentally discovered through an abdominal ultrasound examination that is done for another purpose.
2. A common presentation is the deterioration of health of a known cirrhotic.
3. In areas where HCC is a common tumour an early detection programme is recommended where known cirrhotics have an annual ultrasound examination and alpha-fetoprotein estimation. An elevation of the tumour marker level, or the appearance of a focal lesion warrants further investigations to detect the tumour at its earliest phase.
4. HCC in black Africans grows rapidly ad degenerates producing pain, jaundice, and fever. It also produces a tender mass which is commonly mistaken for an amoebic liver abscesses.
5. Late cases present by pain, jaundice, ascites, hepatomegaly, anorexia, loss of weight and probably massive intraperitoneal haemorrhage.

Investigations

Laboratory tests

1. Serum alkaline phosphatase is often elevated. The rest of liver function tests may show the pattern of cirrhosis if present.
2. Serum alpha-fetoprotein is a tumour marker that is present in the serum of the foetus. The normal adult level is zero to 10 ng/mL. The serum level of this marker is highly elevated with HCC and with testicular teratoma. A level above 200 ng/mL is suggestive, and that higher than 2000 ng/mL is diagnostic of HCC. Other benign liver diseases also elevate the level of serum a-fetoprotein, but to a lesser extent. Serum a-fetoprotein returns to normal level after a successful tumour resection. Re-elevation in the follow up period signifies a recurrence.

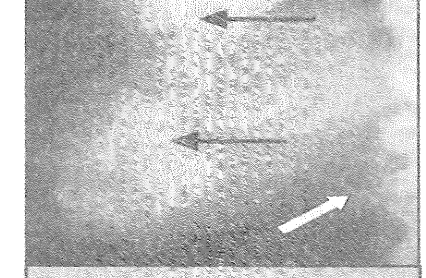


Fig. 31.44. Hepatic angiography. White arrow points at angiography catheter.
Black arrows point at the high vascularity of HCC.

In non-cirrhotics the maximum amount of liver that can be resected is 80%. By time the original mass can be **regenerated**. This figure is much reduced in cirrhotics.

Imaging

1. Ultrasound, CT scan (Fig. 31.43), or MRI can detect the site and extent of tumour spread in the liver. Triphasic (arterial, portal and hepatic) multi-silice CT scan via an i.v. contrast is mandatory to show the anatomical relations of the tumour to the liver vasculature. Preoperative guided biopsy is not desirable in view of the possible risk of haemorrhage from the highly vascular tumour or seeding of tumour cells.
2. Chest x-ray and CT scan to search for chest metastases.
3. Intraoperative ultrasound is used to detect small lesions which may be missed by other imaging modalities. It allows safer liver resection by identification of major vessels and bile ducts.

4. Laparoscopy may be done to visualize the extent of the tumour (peritoneal nodules can be seen) and perform laparoscopic ultrasound.
5. PET-CT scan to detect any systemic metastases.

Treatment

Surgery offers the only hope of cure in patients with HCC. Either hepatectomy or liver transplantation may be performed in suitable patients.

The presence of cirrhosis poses some problems for patients undergoing resection

- Bleeding tendency.
- Poor function of the remaining liver.
- Diminished capacity of liver cell regeneration.

Pre-requisites for hepatectomy

- Child class A patients.
- No evidence of extrahepatic spread.
- No vascular invasion or portal vein thrombosis.
- The tumour is confined to one lobe of the liver.
  The usual operation in such situation is either right or left hemihepatectomy (Fig. 31.45).

Pre-requisites for liver transplantation

- Child class C patients.
- No evidence of extrahepatic spread.
- Single lesion less than 5 cm or 3 small nodules each less than 3 cm (Milan citeria).

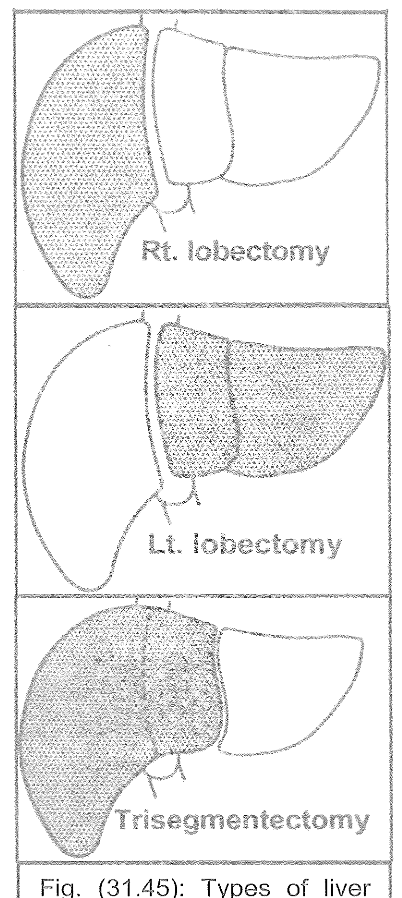


Fig. (31.45): Types of liver resections (continued on next page)

Inoperable cases

The following palliative options are available

- Transarterial chemoembolization **(TACE)** combines the benefits of inducing tumour ischaemia, and those of selective chemotherapy. The chemotherapy agent is loaded on particulate material like gelfoam, and is injected by angiographic techniques into the hepatic artery. The artery is blocked producing tumour ischaemia, and the drug is selectively delivered to its target.
- Systemic chemotherapy.
- Regional ablation by ultrasound guided intra-lesional alcohol, intra-lesional radiofrequency, intra-lesional microwave or laser ablation.

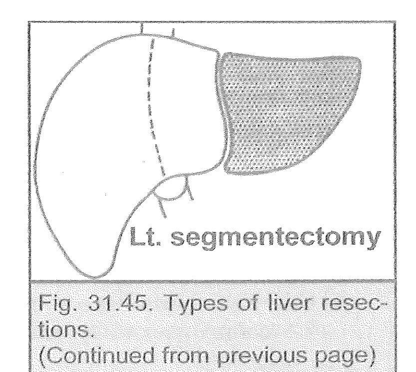


Fig. 31.45. Types of liver resections.
(Continued from previous page)

Liver metastases

Liver Metastases are 20 times more common than primary malignancies.

Sources

Metastases can reach the liver through either:

1. Portal circulation. This is the commonest route. Carcinomas of the colon, rectum, pancreas or stomach metastasize primarily to the liver.

2. Hepatic artery. Carcinomas of the lung, breast, kidney, uterus or ovaries are common examples.
3. Lymphatics.
4. Direct spread from tumours of the gall bladder, stomach or colon.

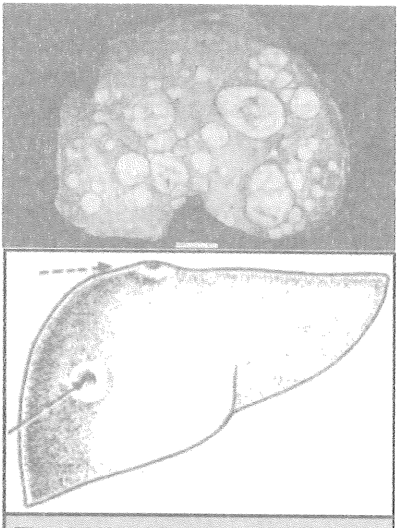


Fig. 31.46. Liver metastases are usually multiple, white and umbilicated.

Pathology

- Metastases may occur in the same time of discovery of the primary tumour (synchronous) or months and years after resection of the primary (meta-synchronous).
- Liver metastatic nodules are usually multiple, white, and umbilicated because of central necrosis (Fig. 31.46).
- They are usually adenocarcinomas.
- Over 90% of patients have tumour deposits in other organs.

Clinical features

In addition to the manifestations that are attributable to the primary tumour, the patient suffers from

1. Weight loss, fatigue, and anorexia.
2. Right upper abdominal pain.
3. Jaundice.
4. Ascites.
5. Palpable hepatomegaly.

Investigations

1. Laboratory tests reveal
   a. Anaemia.
   b. Serum alkaline phosphatase and bilirubin are usually elevated.
2. Imaging by ultrasound, CT scan (Fig. 31.47), or MRI is diagnostic and shows the number as well as the sites of metastases.

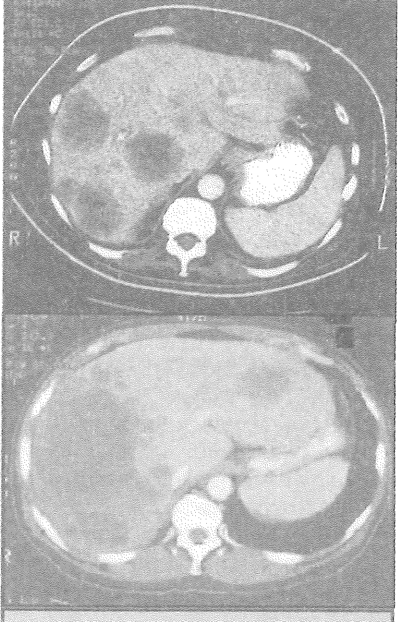


Fig. 31.47. CTs showing multiple liver metastases.

Treatment

Generally, the presence of liver metastases indicates an advanced inoperable tumour. The treatment in such cases, usually by chemotherapy, is considered palliative. A few selected cases of metastatic colorectal cancer, if localized to one segment, can be treated by liver resection aiming at cure.

Chemotherapy

- Systemic chemotherapy is administered in the presence of other organ involvement.
- Selective intra-arterial chemotherapy through a catheter that is surgically inserted in the hepatic artery, is indicated when the liver is found to be the only organ with metastases. This technique reduces the systemic side effects of the anti-cancer agents and allows delivery of higher doses to the liver.

Liver resection

The prerequisites include

1. A completely resectable colorectal cancer with no apparent residual tumour.
2. No extrahepatic metastases are found after a thorough search.
3. Liver metastases that are resectable with a safety margin
    a. A solitary metastatic nodule can be removed in a wedge resection.
    b. Multiple metastases that are confined to one surgical lobe of the liver can be removed by a right or left hemihepatectomy.

Liver resection is done in the same session of the colorectal operation, or later if the liver deposits appear in the course of the patient's follow-up. About 25% of these highly selected patients are cured.

Differential Diagnosis of a focal hepatic lesion

1. Hepatocellular carcinoma.
2. Metastasis.
3. Benign lesion haemangioma, adenoma, and focal nodular hyperplasia.
4. Cyst parasitic or non-parasitic.

The most common

- **Liver tumour is metastatic disease. The ratio of metastasis/primary tumours is 20/1. Primary site is usually the GIT.**
- **Primary malignant liver tumour is HCC.**
- **Benign liver haemangioma hamartoma).**

BILIARY SYSTEM

CHAPTER CONTENTS

Surgical anatomy

The parts of the biliary system are shown on Fig. 32.1.

Gall bladder (GB)

Shape. The gall bladder is pear-shaped, with a normal capacity of about 50 ml.

Location. It is located on the visceral surface of the liver at the plane dividing its two surgical lobes (Fig. 31.5).

Parts. The gall bladder can be divided into fundus, body, infundibulum and neck.

- The fundus is usually located at the angle of the ninth costal cartilage and is completely covered by peritoneum.
- The body of the gall bladder is in contact with the first part of the duodenum and occupies the gall bladder fossa of the liver.
- The infundibulum is the angulated posterior portion of the body of the gall bladder and when dilated it is called Hartmann's pouch.
- The neck forms an S-shaped curve and is connected to the cystic duct.

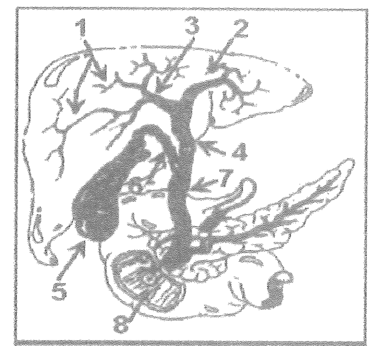


Fig. 32.1. Parts of the biliary system.
1. Intrahepatic biliary radicles.
2. Left hepatic duct.
3. Right hepatic duct.
4. Common hepatic duct.
5. Gall bladder.
6. Cystic duct.
7. Common bile duct.
8. Duodenal papilla.

Extrahepatic biliary tract

Common hepatic duct (CBD)

The common hepatic duct is formed by the union of the right and left hepatic ducts in the porta hepatis.

The cystic duct is 2-3 cm long and 2-3 mm in diameter. Its mucosa is thrown into crescent-like folds called spiral valve of Heister.

Common bile duct

The common bile duct begins at the union of the cystic duct and common hepatic duct and ends at the ampulla of Vater in the second part of the duodenum. It varies in length from 8 to 10 cm and the average diameter is about 6 mm. The common bile duct can be divided into four parts; supraduodenal, retroduodenal, intrapancreatic and intra-duodenal.

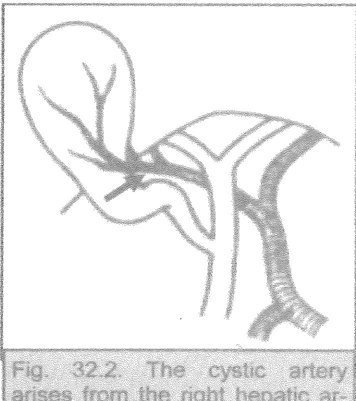


Fig. 32.2. The cystic artery arises from the right hepatic artery.

The supraduodenal part is about 2.5 cm long, and runs in the free margin of the lesser omentum (Fig. 31.3). The intraduodenal part passes through the wall of the second part of the duodenum, where it joins the main pancreatic duct, and together form a dilated segment that is known as the ampulla of Vater. The latter opens in the middle of the

second part of the duodenum on the major duodenal papilla. The common opening is surrounded by the sphincter of Oddi.

Blood supply

Arteries

- The gall bladder is supplied by the cystic artery, which usually arises from the right hepatic artery (Fig. 32.2).
- The extrahepatic bile ducts are usually supplied from the cystic artery above and gastroduodenal artery below. The blood vessels supplying the ducts usually run longitudinally at the 3 and 9 o'clock positions.

Veins

Venous return consists of small veins that directly enter the liver bed and of a cystic vein that enters the right branch of the portal vein.

Lymphatic drainage

The lymph vessels of the gall bladder drain into the cystic lymph node of Lund which lies in front of the junction of the cystic and common hepatic duct.

Efferent vessels from this lymph node pass to the hilar and to the coeliac lymph nodes. The subserosal lymphatics of the gall bladder connect with the subcapsular lymphatics of the liver.

Nerve supply

Parasympathetic (vagal) fibres arise from the hepatic division of the anterior vagal trunk. Parasympathetic supply is responsible for contraction of the gall bladder. Its effect on the sphincter of Oddi is variable. Sympathetic fibres reach the celiac plexus by way of the greater splanchnic nerves. Then sympathetic fibres reach the gall bladder along the blood vessels, Sensory supply is transmitted along the splanchnic nerves to the 7-10th thoracic nerves.

Surgical physiology

Bile secretion

Amount

Normal adults secrete 250-1000 ml bile per day.

Control

- Neurogenic. Vagal stimulation increases secretion; splanchnic stimulation decreases bile flow.
- Hormonal. Bile flow is stimulated by secretin release from the duodenum which itself is stimulated by hydrochloric acid, protein breakdown products and fatty acids.

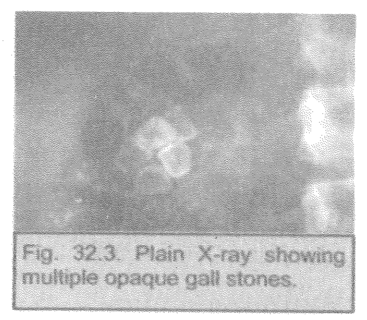
Composition of bile

The main constituents of bile are:

1. Water.
2. Electrolytes in the same concentration as the plasma.
3. Bile salts. The major bile acids which form these salts are cholic, deoxycholic, and chenodeoxycholic acids. These conjugate with taurine or glycine.
4. Cholesterol and phospholipids. Cholesterol is insoluble in water and is carried in bile by the combined detergent effect of bile salts and phospholipids.
5. Bile pigments. The yellow colour of bile is due to the presence of the pigment bilirubin diglucuronide, which is the break down product of haemoglobin.

Gall bladder function

1. The gall bladder **stores and concentrates bile**. Sodium, chloride, and water are selectively absorbed, resulting in 10-fold increase in concentration of bile salts, bile pigments and cholesterol.
2. **Mucus secretion** protects the mucosa from the lytic action of bile and facilitates passage of bile through the cystic duct.
3. Gall bladder contracts and empties its content when bile is needed for digestion. Its emptying is mediated by both humoral and nervous stimulation. Cholecystokinin is released from intestinal mucosa in response to food and is responsible for contraction of the gall bladder and relaxation of the sphincter of Oddi. It leads to evacuation of 70% of the gall bladder contents within 30 minutes. Vagal innervation stimulates contraction, whereas sympathetic stimulation inhibits motor activity.


Fig. 32.3. Plain X-ray showing multiple opaque gall stones.

Imaging investigations

Plain x-ray

This test is of a limited value.

- It can detect gall stones in only 10-15% of cases (Fig. 32.3).
- A gall bladder with a calcified wall (porcelain) can be visualized.
- Presence of air in the gall bladder can be seen in acute emphysematous cholecystitis, while air in the biliary system is present after choledochoduodenostomy or sphincterotomy operations.

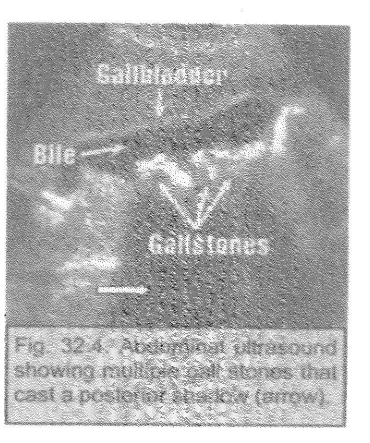


Fig. 32.4. Abdominal ultrasound showing multiple gall stones that cast a posterior shadow (arrow).

Abdominal ultrasound

This is now the first investigation to be performed in most disorders of the biliary system.

Advantages. It is easy, simple, inexpensive and non invasive investigation. It can be performed in the presence of acute inflammation or jaundice.

It can provide the following information:

- Detection of gall bladder stones in a 98% of cases (Fig. 32.4). It is less accurate in the detection of stones of CBD.
- Detection of the thickness of the wall of the gall bladder.
- Visualization of dilatation of extra and intrahepatic biliary ducts in patients with surgical obstructive jaundice.
- Detection of masses in the porta hepatis, or head of pancreas.

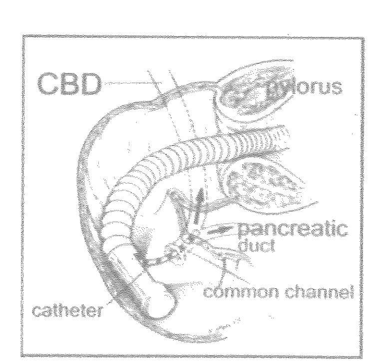


Fig. (32.5) ERCP technique

Oral cholecystography:

This was previously the investigation of choice for detection of gall stones, but now it has been replaced by ultrasonography.

Endoscopic retrograde cholangiopancreatography (ERCP)

Method (Fig. 32.5)

An endoscope is passed and the oesophagus, stomach and duodenum are inspected. The duodenal papilla is localized and a cannula is passed in it and a contrast material is injected to visualize the common bile and the pancreatic duds by radiography.

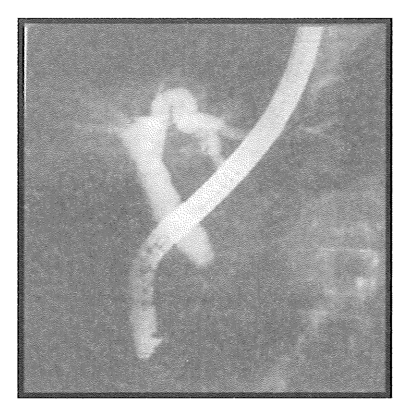


Fig. (32.6) ERCP showing carcinoma of the head of the pancreas

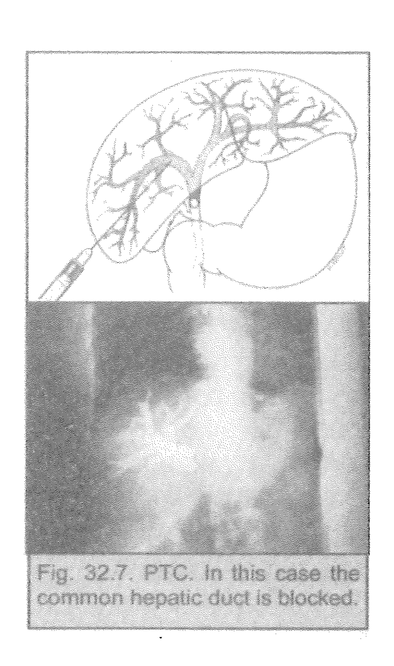


Fig. 32.7. PTC. In this case the common hepatic duct is blocked.

Indications

ERCP is very valuable in the following situations

- Diagnosis of lesions involving the lower end of CBD as carcinoma of head of pancreas or ampulla of Vater. A biopsy can be taken from an ampullary lesion (Fig. 32.6).
- Detection of missed calculi in the CBD after cholecystectomy.
- Detection of operative injuries of the biliary system.
- Visualization of the pancreatic duct in patients with chronic pancreatitis or pancreatic pseudocyst.
- Therapeutic procedures can be performed with ERCP, e.g.,
  - Sphincterotomy and removal of bile duct stones.
  - Insertion of a stent to drain malignant obstruction of the duct.

Complications

In the presence of unrelieved obstruction in the CBD, injection of the contrast material may lead to cholangitis or even to septicaemia due to increased pressure in of the CBD. Acute pancreatitis or duodenal perforation may occur.

Percutaneous transhepatic cholangiography (PTC)

Pre-requisites

1. Normal coagulation. The prothrombin time (PT) and concentration are checked. If PT is prolonged, vitamin K is given I.V for a few days to correct hypo-prothrombinaemia before the procedure.
2. Dilated intrahepatic biliary radicles, as seen on ultrasound.

Technique (Fig. 32.7)

Under local anaesthesia, a thin cannula (Chiba needle) is introduced through the 8th intercostal space into the liver and continuous suction by a syringe is applied until bile is aspirated and the dye is then injected. The dye will visualize the intrahepatic biliary ducts and then it will flow to the bile duct from above downwards.

Indications

PTC is particularly indicated to diagnose high obstruction of bile ducts as in post-operative biliary strictures or in hilar cholangiocarcinoma.

Complications

- Bleeding may occur if there is hypoprothrombinaemia.
- Biliary peritonitis.

Radioisotope scanning

Method

^{99m}Tc labelled derivative of iminodiacetic acid (HIDA) is injected LV. The isotope is rapidly excreted by the liver to visualize the biliary tree.

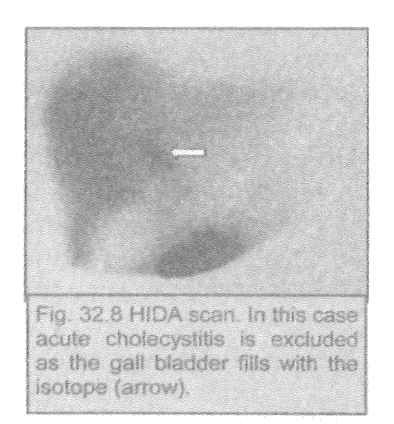


Fig. 32.8 HIDA scan. In this case acute cholecystitis is excluded as the gall bladder fills with the isotope (arrow).

Uses

- Diagnosis of acute cholecystitis. Non-visualization of the gall bladder indicates obstruction of its neck and supports the diagnosis. A gall bladder that fills by the isotope excludes it (Fig. 32.8).
- Diagnosis of congenital biliary atresia.
- To visualize a biliary enteric anastomosis.



Magnetic resonance cholangiopancreatography (MRCP)

MRCP is expensive but can provide good quality images without the complications associated with ERCP or PTC (Fig. 32.9)

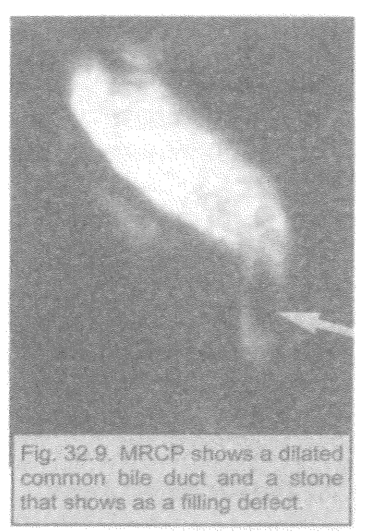


Fig. 32.9. MRCP shows a dilated common bile duct and a stone that shows as a filling defect.

Congenital anomalies

Congenital anomalies of the biliary tract are found in 10 percents of autopsies.

Anomalies of the gall bladder

- Absence of the gall bladder is rare; occurring in 0.03% of cases.
- The phrygian cap is kinking of the fundus of the gall bladder and is present in 2 to 6 percent of cholecystographies. It is of no clinical significance.
- Double gall bladder with two separate cavities and cystic ducts occurs in 1 in 4000.
- Floating gall bladder. The organ hangs on a mesentery which makes it liable to undergo torsion.
- Left sided gall bladder is extremely rare.
- Intrahepatic gall bladder can occur and is associated with an increased incidence of gall stones.

Anomalies of bile ducts

Biliary atresia

Incidence. 1 per 10.000 live births.

Aetiology is unknown but may be the result of an inflammatory process which results in occlusion of variable lengths of biliary tree.

Types

- Correctable type. There is a patent portion of the extrahepatic duct which communicates with the intrahepatic ducts. This type is present in only 10% of patients.

- Non correctable type. The extrahepatic portion of the biliary tract is occluded and appears as a cord like structure. This type unfortunately represents 90% of cases.

Clinical Picture. Jaundice is present since birth, but may not be marked until after the first several weeks. Urine becomes dark and the stools are pale. The abdomen may gradually become distended by the enlarging liver or by ascites. Eventually, the spleen also enlarges.

Diagnosis

- Serum bilirubin is elevated and the direct fraction is at least half of the total.
- Radioisotope scanning. If there is no excretion of the radionuclide into the intestinal tract, atresia is virtually assured.
- Laparotomy is needed to confirm the diagnosis.

Differential diagnosis

This includes physiological jaundice, haemolytic diseases, neonatal hepatitis, alpha$_1$-antitrypsin deficiency, inspissated bile syndrome, infection with different viruses or metabolic defects.

Treatment

- Correctable type. A Roux-en-Y jejunal loop is anastomosed to a patent portion of the extrahepatic biliary tract.
- Non correctable type. This type of lesion is treated by an operation called portoenterostomy (Kasai operation) in which the occluded extraheaaptic biliary tree is excised up to the liver capsule and a Roux loop of jejunum is anastomosed to this area to drain bile from minute biliary channels. If portoenterostomy fails, liver trans-plantation becomes the treatment of choice.

Choledochal cyst

Pathology

This is a rare congenital anomaly in which there is dilatation of the common bile duct of unknown cause.

The condition is more common in females and it leads to dilatation of the intra and/or extrahepatic biliary tree. Diffuse fusiform dilatation is the most common type (Fig. 32.10).

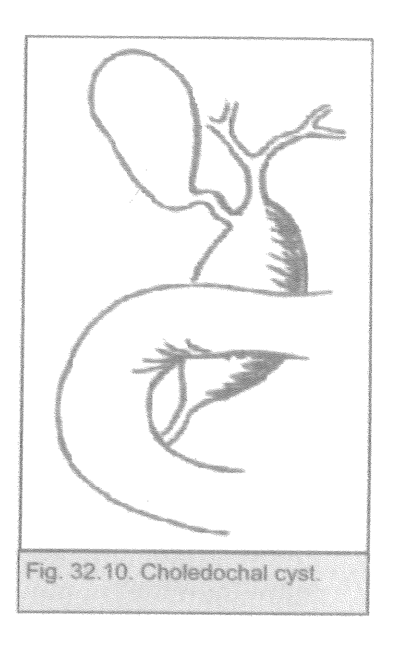


Fig. 32.10. Choledochal cyst.

Clinical features

The majority of cases are diagnosed in childhood before 10 years of age. Obstructive jaundice, pain and right hypochondrial mass are the main clinical presentations. Recurrent attacks of pyrexia due to cholangitis may occur.

Treatment

The cyst should be excised and choledochojejunostomy is performed.

Hepatic duct anomalies

- Accessory hepatic duct may enter into the cystic duct or into the neck of gall bladder.
- The right, left or even both hepatic ducts enter the gall bladder. Ligation of these ducts during cholecystectomy could result in a surgical catastrophe.

Cystic duct anomalies

- Absence of the cystic duct. Injury of the common bile duct is liable to occur when cholecystectomy is performed in a patient with this abnormality.
- Low insertion of cystic duct. The cystic duct opens into the common duct near the ampulla. This anomaly is common and it may lead to ligation of the common bile duct during a cholecystectomy operation.
- The cystic dud may enter the right hepatic duct and the latter may be mistaken for the cystic duct and ligated.

Anomalies of the hepatic and cystic arteries

- Accessory cystic artery is the most common anomaly and may be torn if it is not identified.
- A large accessory left hepatic artery may arise from the left gastric artery in about 5% of people.
- The right hepatic artery may arise from the superior mesenteric artery.
- The right hepatic artery may pass in front of or behind the common hepatic duct or common bile duct and may be mistaken for the cystic artery and is, therefore, ligated.

Definitions

Cholelithiasis
Gall bladder stones
Choledocholithiasis
Common bile duct stones
Cholecystitis
Inflammation of gallbladder
Cholangitis
Infection of bile ducts

Gallstones

Incidence

- There is a geographical variation in the incidence of gall stones, being more common in Western countries. This may be related to dietary factors. In these countries they occur in at least 20% of women over the age of 40.
- A female-to-male ratio of 3:1 is reported in cholesterol and mixed stones. The increased incidence in females is not present after the age of 60. Female preponderance is not present with pigment stones.
- The incidence of gall stones increases with age.
- There is also familial incidence of the disease, as it is more common in parents and siblings of patients than in matched control subjects.

cholesterol

mixed

pigment

Fig. 32.11. Types of gall stones

Types and composition (Fig. 32.11)

- **Pure cholesterol stones** are usually single (cholesterol solitaire), yellow, more than 2.5 cm in diameter and contain nearly 100% of their weight cholesterol. These stones are radiolucent. Sometimes there are multiple cholesterol stones.
- **Mixed stones** are usually multiple, yellowish, faceted, range from 0.5-2.5 cm in diameter and contain more than 60% of their weight cholesterol. In addition they include calcium bilirubinate, calcium phosphate and calcium palmitate. This type accounts for 90% of all calculi. The stones are radio-opaque in 15% of cases.
- **Pigment stones**
  - **Black pigment stones**. These are usually multiple, tarry black with multiple spicules. The stones are less than 2.5 cm. They are composed of calcium bilirubinate, and are radio-opaque in 50% of cases. These stones are found in patients with haemolytic anaemias or cirrhosis.
  - **Brown pigment stones** These are multiple, brown, laminated and less than 2.5 cm in diameter. They form in infected, stagnant bile, usually in the bile

duct, or in the presence of foreign bodies, e.g., a biliary stent. They are radio-opaque. The major constituent is calcium bilirubinate, but calcium palmitate and cholesterol may be present.

Aetiology

Cholesterol and mixed gall stones

- **Disturbed bile salts cholesterol ratio**. Cholesterol is virtually insoluble in water and is carried in bile by the combined detergent effect of bile salts and phospholipids which form micelle aggregates with it. A certain ratio (25:1) between bile salts and phospholipids on one hand and cholesterol on the other hand has to be maintained to keep cholesterol in solution. Any lowering of this ratio can lead to supersaturated bile (lithogenic bile) with consequent cholesterol precipitation. The following factors may disturb this ratio:
    - Reduced bile salt pool.
        - Malabsorption of bile salts in the terminal ileum in Crohn's disease, small bowel resection or bypass procedures. In diabetes mellitus autonomic neuropathy of the ileum may affect the enterohepatic circulation and the bile acid pool.
        - Diminished hepatic synthesis in liver disease.
        - Estrogens reduce the concentration of bile salts in bile.
    - Increased cholesterol synthesis. This may occur in obesity, high dietary fat, and high caloric diet.

- **Stasis of bile:**
    - Female hormones. Progesterone causes relaxation and impaired emptying of the gall bladder. Oestrogen, oral contraceptives and repeated pregnancy are associated with increased incidence of gall stones.
    - Following truncal vagotomy due to denervation of the gall bladder.
    - Diabetes mellitus.
    - Obesity.
    - Long term parenteral nutrition due to lack of oral intake which precludes release of cholecystokinin.

Pigment stones

- Haemolytic anaemias. Any condition which shortens the life span of red blood cells, as hereditary spherocytosis, thalassaemia or prosthetic heart values may be associated with black pigment stones.
- Liver cirrhosis is associated with an increased incidence of gall stones overall, but black pigmented stones are the commonest. The cause of pigment stone formation is most probably due to decreased secretion of bile acids by the cirrhotic liver leading to diminished solubility of any unconjugated bilirubin.
- Infection plays a role in the formation of brown pigment stones. Infection by some strains of E. coli leads to the production of B-glucuronidase enzyme which hydrolyses bilirubin glucuronide into the insoluble bilirubin which precipitates as calcium bilirubinate.

Most gallstones are silent and remain so in 70% of patients, being diagnosed as an incidental finding, usually, on ultrasound. Symptoms (recurrent biliary colic) develop in 2-3% per year. Each year, 3-5% of symptomatic patients develp complications. Thus prophylactic cholecystectomy for silent stones is rarely indicated.

Complications (Fig. 32.12)

Complications in the gall bladder

- Obstruction of the cystic duct or neck of gall bladder.
    - Mucocele of the gall bladder if the contents remain sterile.
    - If infected the result is acute calcular cholecystitis with or without empyema, gangrene or perforation.
- Chronic calcular cholecystitis. The gall bladder is usually thick-walled and is commonly contracted.
- Carcinoma of the gall bladder. A serious complication of long-standing calcular disease is the development of squamous metaplasia of the gall bladder followed by carcinoma. About 90% of patients with carcinoma of the gall bladder have gallstones.

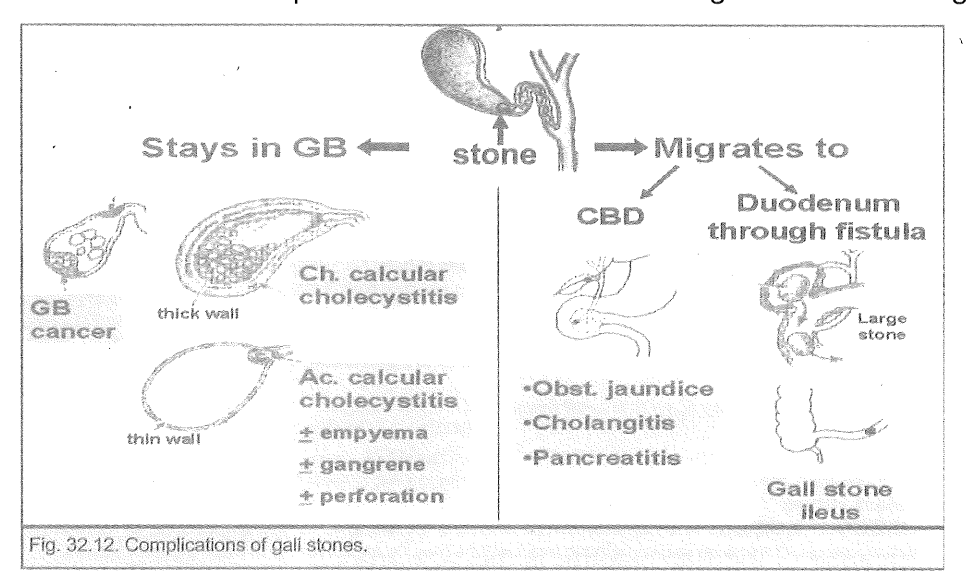


Fig. 32.12. Complications of gall stones.

Complications caused by migration of stones

- **Migration to CBD**
    - **Obstructive jaundice**, with its consequences; bleeding tendency, septicaemia, renal and hepatic failure.
    - **Cholangitis** and cholangitic abscesses of the liver.
    - **Acute pancreatitis** occurs if a stone in the lower end obstructs the common channel of CBD and pancreatic duct. It is to be stressed that in many cases of calcular pancreatitis, there is no actual obstruction of the ampulla by a stone during the attack and it has been suggested that the mere passage of a stone through the ampulla may initiate an unknown mechanism which precipitates pancreatitis (Chapter 33).
    - Rarely, in long-standing intermittent obstruction, **biliary cirrhosis** results.

- **Migration to duodenum through a fistula**
    - **Gallstone ileus** is a rare complication. A big stone (> 2.5 cm) that passes through a fistula between the gall bladder and duodenum may get impacted in the terminal ileum about two feet from the ileocaecal valve. This form of intestinal obstruction is called gallstone ileus obstruction. Clinical diagnosis is usually difficult as the patient is often an elderly and gives a long history of biliary problems. The symptoms of intestinal obstruction are usually misinterpreted as being due to biliary problems (Chapter 36).

Clinical features

A patient with gall stone may present by one of the following:

1. **Accidental discovery of silent gallstones**. Plain X-ray of the abdomen or ultrasound performed for an abdominal problem may detect silent gallstones. Most of gallbladder stones are silent.
2. **Recurrent attacks of biliary pain**. The usual complaint of most patients is recurrent attacks of severe upper abdominal pain which may be referred to the back of the right chest (Fig. 32.13) or right shoulder. The pain is constant, increases in severirty over 30 minutes and lasts less than 5-6 hours, longer duration suggests acute cholecystitis. Pain is usually precipitated by the intake of a fatty meal. There may be nausea or vomiting during the attack. Clinical examination may reveal tenderness in the right hypochondrium. Murphy's sign may be positive (the gall bladder area is palpated while the patient is asked to take a deep breath; the patient will catch her breath). Sometimes, these patients are diagnosed as having peptic ulcer or chronic colitis.

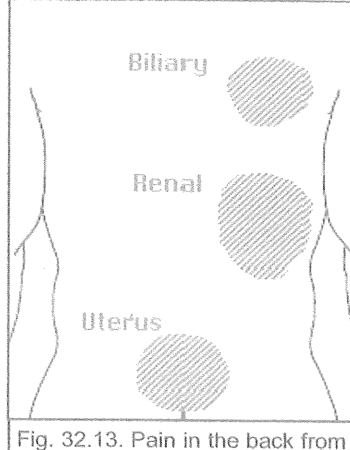


Fig. 32.13. Pain in the back from different sources.

3. Biliary dyspepsia. A common complaint is the occurrence of fatty dyspepsia with bloating and excessive eructations following fatty meals.
4. Reflex symptoms. Gallstones may cause reflex retrosternal pain which is usually diagnosed as anginal pain. Actual ECG changes may occur.
5. Complications. In some patients the first evidence of calcular disease of the gall bladder is the occurrence of a complication, e.g., obstructive jaundice.

> **Ultrasound**
>
> U/S can see almost all gall bladder stones (98%).
>
> U/S can see only one third of CBD stones.

Investigations

- **Plain x-ray of** the upper abdomen. This has a very low practical value as only 10-15% of gall stones are radio-opaque (contrary to renal stones). Opaque gall stones are usually multiple, faceted, signet ring (Fig. 32.3) and if a lateral view is taken they are located anterior to the spine.
- **Abdominal ultrasound**. This simple, inexpensive and non- invasive technique is now the investigation of choice. It can provide the following information:
    - Detection of gall stones in 98% of cases Fig. 32.4 & 32.14).
    - Thickness of the gall bladder wall. A thickened wall denotes long-standing recurrent inflammations.
    - The diameter of the common bile duct and any intrahepatic biliary dilatation which direct attention to the presence of a stone in the common bile duct.

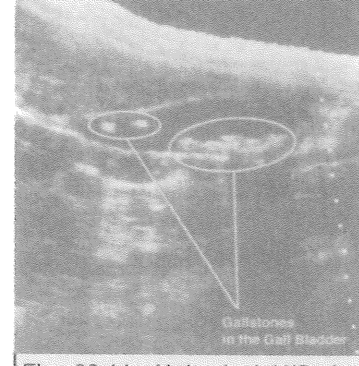


Fig. 32.14. Abdominal U/S detects gallbladder stones in 98% of cases.

Treatment

The standard treatment of gallstones is cholecystectomy, which may be either done by the open method or by the laparoscope. Extracoporeal shock wave lithotripsy (ESWL),

and medical dissolution of gallstones had some popularity in the 1980s. Because of their limited efficacy and because of their many side effects, they have dropped out of use.

What to do for patients with silent gallstones?

Prophylactic cholecystectomy is rarely indicated for silent stones since they remain asymptomatic in some 70% of people over 20 years. It may be advisable in elderly diabetics (increased mortbidity and mortality if acute cholecystitis develops). A definite indication is gallbladder wall calcification (porcelain gallbladder) which is precancerous.

Acute cholecystitis

In the majority of cases (about 98%) acute cholecystitis is secondary to obstruction of the cystic duct or Hartmann's pouch by a stone.

Fig. 32.16. Acute calcular cholecystitis is caused by stone obstruction of the gallbladder neck or the cystic duct.

Acute calcular cholecystitis

Pathology

Pathogenesis

Obstruction of the cystic duct or neck of gall bladder by a stone and consequent infection, usually by Gram negative bacilli, result in acute cholecystitis (Fig. 32.16).

Gross appearance

The gall bladder is distended, its wall is thickened, and its serosal lining loses its normal bluish lustre and is covered by fibrinous deposits. Multiple microabscesses occur in the wall. As a defensive mechanism, the greater omentum, the duodenum and colon become adherent to the gall bladder to localize infection.

Why is acute appendicitis treated by surgery while acute cholecystitis may be treated conservatively?

1. The organisms causing acute appendicitis are much more virulent and abundant than those causing acute cholecystitis.
2. The appendix has only the appendicular artery for its supply, while the gall bladder has numerous vascular connections to the undersurface of the liver in addition to the cystic artery. Thrombosis of the appendicular artery will lead to gangrene of the appendix.

Consequences

- Resolution. In most cases, with treatment, the stone dislodges and the obstruction is relieved with gradual resolution of inflammation.
- Less commonly obstruction persists with progressive distension of the gall bladder. Thrombosis of the blood vessels may occur and patches of gangrene may develop in the wall. Perforation of the gall bladder may occur followed by either localized or less commonly generalized peritonitis (0.5%). A special form called "acute emphysematous cholecystitis" may occur in diabetic patients due to infection by anaerobic organisms, e.g. clostridia. The infection is highly virulent, is accompanied by gas formation and leads to early gangrene of the gall bladder.
- Empyema of gall bladder. The gallbladder is converted into a closed bag of pus.
- Recurrent attacks lead to chronic calcular cholecystitis.

Clinical features

Symptoms

- Initially the patient complains of diffuse colicky upper abdominal pain (similar to biliary colic). With inflammation of the serosal covering, pain becomes dull aching, persistent and localizes in the right hypochondrial region. Pain may be referred to the right shoulder due to irritation of the undersurface of the diaphragm supplied by sensory

fibres of the phrenic nerve. Pain persisting more than 6 hours usually denotes acute cholecystitis.
- There is usually nausea, or sometimes vomiting.

Examination
- Usually there is pyrexia and tachycardia.
- Abdominal examination reveals tenderness, rebound tenderness and muscle guarding in the right hypochondrium.
- It is difficult to palpate the distended gall bladder due to the overlying tenderness and rigidity.
- Sometimes, an area of hyperaesthesia may be elicited between the 9th to the 11th ribs posteriorly on the right side (Boas's sign).
- Rarely, a large stone impacted in the Hartmann's pouch may compress the bile duct leading to jaundice (Mirrizi syndrome).

Differential diagnosis
1. Perforated duodenal ulcer,
2. Acute pancreatitis.
3. High retrocaecal appendicitis.
4. Right pyelonephritis.
5. Amoebic hepatitis.

Investigations
Laboratory
- Blood picture. Polymorphonuclear leucocytosis may be present.
- Liver function tests are usually normal.

Imaging
- Abdominal ultrasound. This is the most appropriate investigation. It will reveal the presence of gall stones, distended gall bladder, thickened wall and it can also reveal microabscesses and serosal oedema due to spreading inflammation.
- HIDA scan. ^{99m}Tc is administered Iv. It is secreted by the liver, and is then excreted along the biliary pathway. Normally it is concentrated in the gall bladder. If the common bile duct is visualized while the gall bladder is not seen, this is diagnostic of acute cholecystitis. This investigations is not commonly performed.

Treatment
There are two options, but ultimately the definitive treatment is cholecystectomy.

- **Early cholecystectomy within 3 days of** onset of attack. So long as the diagnosis is confident, the **patient is** fit and the surgeon is experienced, early cholecystectomy is now recommended. Proponents of this line of treatment claim that it has the following advantages
  - Early surgery (within 3 days) is not unduly difficult.
  - It avoids the complications of acute cholecystitis that may arise during conservative treatment.
  - One hospital admission with early return to work.

- **Initial conservative treatment followed by cholecystectomy after 6 weeks**
  - Nil by mouth, and IV fluids.

- Antibiotics effective against gram negative bacilli as Ampicillin or a cephalosporin.
- Analgesics as pethidine or NSAIDs.
- Follow-up of the pulse, temperature, area of tenderness and rigidity.
- Under this treatment, most patients improve and the attack subsides. The patient is sent home, and is advised to come back after 6 weeks to have elective cholecystectomy. This long delay is to allow the adhesions to subside.
- If on conservative treatment, the patient deteriorates, surgery should be performed
  - Cholecystectomy if safely feasible.
  - Cholecystostomy is performed if dense adhesions make cholecystectectomy a risky operation. The gall bladder fundus is opened and the stones are removed. The gall bladder is drained by a tube for one week (Fig. 32.17). The patient usually returns for elective cholecystectmy after 6 weeks.

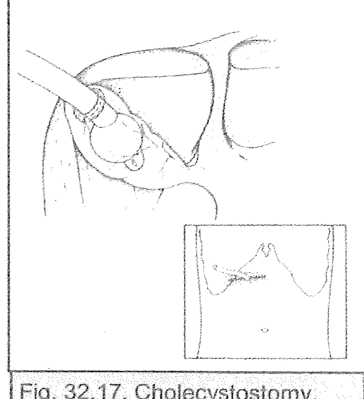


Fig. 32.17. Cholecystostomy.

- Advocates of conservative treatment claim the following reasons for this policy:
  - Most of the patients will settle on conservative treatment.
  - Surgery in the acute stage is difficult as the tissues are friable, oedematous and there are adhesions making identification of important structures difficult.

  N.B. It is the trend now to treat acute cholecystitis by early surgery).

Acute non-calcular cholecystitis

- This is a rare and serious condition which may occur in
  - Patients suffering from major burns or major trauma.
  - Patients on prolonged total parenteral nutrition.
  - It may complicate certain infections as brucellosis and typhoid.

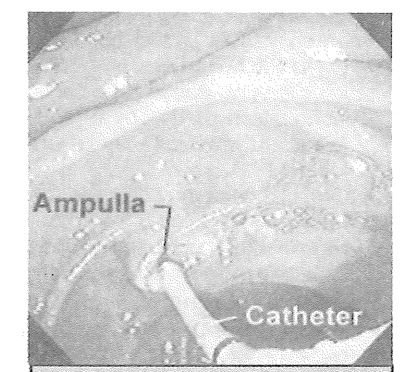
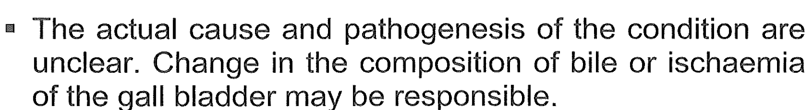


Fig. 32.18. Endoscopic view of the duodenum showin cannulation of duodenal papilla for ERCP.

- The actual cause and pathogenesis of the condition are unclear. Change in the composition of bile or ischaemia of the gall bladder may be responsible.



- The clinical picture is similar to that of acute calculous cholecystitis, but usually the diagnosis is delayed because it is not suspected.
- Ultrasound examination is diagnostic.
- Treatment is by urgent cholecystectomy.

Common bile duct stones

Incidence

About 15% of patients with calcular cholecystitis have stones in the CBD. Bile duct stones are called choledocholithiasis.

Pathology

Formation

- In most cases, a stone in the common bile duct (CBD) is originally formed in the gall bladder, and has slipped through the cystic duct into the CBD.
- Primary stones in the CBD are rare and occur when there is prolonged stasis and infection; these stones have the chemical composition of brown pigment stones.

Sequelae

1. No effect if the stone remains floating in CBD without obstruction.
2. Passage to the duodenum. A small stone, usually less than 3mm, can pass spontaneously through the sphincter of Oddi. During its passage it may produce acute pancreatitis.
3. Obstruction of CBD.
   a. Obstructive jaúndice.
   b. Dilatation of the bile duct and intrahepatic biliary radicles,
   c. In the presence of prolonged obstruction and high pressure in the bile ducts, bile secretion by the liver stops. The bile ducts become full of mucous (white bile).
   d. Complications
      i. Bleeding tendency. Bile salts fail to reach the bowel, with failure of absorption of fat-soluble vitamins including vitamin K. Consequent deficient synthesis of coagulation factors occurs.
      ii. Cholangitis. Presence of calculi in the biliary pathway predisposes to the development of recurrent attacks of cholangitis due to infection by Gram-negative bacilli. Cholangitis leads to the development of severe infection of the liver or even septicaemia.
      iii. Septicaemia and hepatorenal failure. Absence of bile salts in the intestine allows absorption of bacterial endotoxins which may fail to be filtered by Kupffer cells, Endotoxins are supposed to be responsible for the renal failure which may occur in these patients. They cause renal vasoconstriction and are associated with glomerular and peritubular fibrin deposits. Shortly liver failure follows. Septicaemia and hepatorenal failure are the usual causes of death in unrelieved CBD obstruction.
      iv. Acute pancreatitis occurs if the stone impacts in the lower part at the common channel of pancreatic duct and CBD (Fig. 334).
      v. Biliary cirrhosis is uncommon. It occurs in prolonged cases where obstruction is intermittent or incomplete.

Clinical picture

Symptoms

1. **Silent stones**. If there is no obstruction, there are no symptoms.
2. **Pain**. The patient may complain of recurrent attacks of severe dull aching pain in the right hypochondrial and epigastric regions which may be referred to the right scapular region or even to the back. During the attacks there may be nausea and vomiting.
3. **Jaundice**. Calcular obstructive jaundice is usually slowly progressive, fluctuating and usually does not reach a severe degree. It is due to impaction of the stone in the narrow distal part of the CBD. It is to be noted that there is no relation between the size of the stone and the development of jaundice. A tiny stone impacted in the ampulla may cause jaundice, while a large calculus in a dilated CBD may not cause obstruction. After a while an impacted stone either slips to the duodenum or floats

back up to the dilated CBD with relief of jaundice. Calcular jaundice is obstructive in nature with dark frothy urine, pale stools and pruritus.

4. **Charcot** triad due to superadded cholangitis, the patient gives a history of recurrent attacks of:
    a. Pain
    b. Jaundice
    c. Fever and rigors.

    Reynold's pentad
    a. Charcot triad
    b. Altered mental status
    c. Shock

Examination

1. Degree of jaundice.
2. Temperature.
3. Signs of hepatocellular failure.
4. Signs of bleeding tendency.
5. Itching marks.
6. **The gall bladder is usually not palpable** since its wall is fibrosed and thickened due to long-standing chronic calcular cholecystitis; resisting the pressure of retained bile. Calcular obstruction is also characteristically intermittent leading to a lower rise in the intrabiliary distending pressure than in malignant obstructive jaundice. Malignant obstruction due to carcinoma of the head of pancreas results in almost complete continuous obstruction leading to a much higher rise in pressure, which distends a healthy thin-walled gall bladder resulting in a large palpable gall bladder, which is easily felt clinically. Therefore, a palpable distended gall bladder in a patient with obstructive jaundice is unlikely to be caused by stone obstruction (Courvoisier's law, Fig. 33.12).

Differential diagnosis of calcular obstructive jaundice

- Hepatocellular jaundice due to:
    - Viral hepatitis.
    - Drug toxicity.
- Malignant obstructive jaundice.
    - Carcinoma of head of pancreas (Chapter 33 and table 33.2).
    - Cholangiocarcinoma of bile ducts.

Investigations

Laboratory

Blood Picture. During an attack of cholangitis, polymorphonuclear leucocytosis may occur.

Liver function tests

1. Raised bilirubin level; mainly the direct fraction. Usually bilirubin does not exceed 10 mg/dL and its level may fluctuate.
2. Alkaline phosphatase If other causes of raised alkaline phosphatase are excluded, this enzyme is the most sensitive indicator of biliary tract obstruction.



Fig. 32.19. ERCP shows an enlarged CBD with two filling defects inside. These are two unusually large stones.

3. Prothrombin time and concentration are disturbed due to failure of absorption of vitamin K.
4. Serum aminotransferases; ALT (SGOT) and AST (SGPT) may show slight rise, especially if there is cholangitis.
5. Gamma glutamyl transferase and 5-nucleotidase are both maximally elevated in obstructive jaundice.
6. Faecal stercobilinogen and urinary urobilinogen are diminished.

Imaging

- **Abdominal ultrasound**. Provides the following information:
  - Dilatation of intrahepatic biliary radicles in patients with obstructive jaundice.
  - The diameter of the CBD (normal 4 to 7mm) is increased.
  - Presence of chronically inflamed gall bladder with calculi points to but is not conclusive evidence that obstruction is due to calculi.
  - Presence of stones in the CBD in a good number of cases.
  - A mass in the head of the pancreas may be detected.
- **Abdominal CT** is valuable in demonstrating lesions of the pancreas or metastatic lymph nodes.
- **ERCP** (Fig. 32.18) can detect lesions of the ampulla and a biopsy can be taken. Both the CBD and pancreatic ducts will be visualized. A stone will appear as a filling defect in the CBD (Fig. 32.19). ERCP should not be done during an attack of cholangitis unless a therapeutic procedure to drain bile is planned because the rise of pressure during injection of the contrast material may lead to severe systemic sepsis.
- **MRCP**

Management of calcular obstructive jaundice

Aim

1. To relieve biliary obstruction by removal of stones from CBD.
2. To remove the gall bladder, that is usually the source of CBD calculi.

Preoperative preparation

In the absence of cholangitis, calcular obstructive jaundice is not an emergency. The patient can be admitted to hospital for a few days during which he receives

1. Injections of vitamin K1 to correct the coagulation abnormalities.
2. Adequate hydration by IV fluids to prevent the possibility of renal failure. IV mannitol may be prescribed if urine output is not satis factory
3. Oral bile salts may diminish the liability to endotoxaemia and Thenal failure.
4. Broad spectrum antibiotics if there is evidence of cholangitis.

Fig. 32.20.
A. Endoscopic sphincterotomy.
B. Stone removal by Dormia basket.

Definitive treatment

- If the patient is debilitated with suppurative cholangitis, temporary relief of biliary obstruction can be achieved by PTD or stent insertion at ERCP and definitive management postponed until the patient is fit for surgery.
- Treatment is indicated for both silent and asymptomatic common bile duct stones.

- **If ERCP is available**, the favoured treatment is endoscopic extraction of calculi followed by cholecystectomy (open or by laparoscopy). Avoidance of CBD exploration at cholecystectomy reduces operative and postoperative complications.
  - Procedure (Fig. 32.20).
    - i. Diathermy (electrocautery) is used to do endoscopic sphincterotomy.
    - ii. The stone(s) is removed by Domlia basket or a balloon catheter.
    - iii. A large stone can be fragmented before removal. This is done either by mechanical, electrohydraulic, or laser lithotripsy.
  - Possible complications
    - i. Bleeding occurs in 2-9% of cases. This may be due to coagulation defects. The prothrombin time should be checked before the procedure and vitamin K administered IV.
    - ii. Acute cholangitis occurs in 1-3% of cases. It may progress to septicaemia and death especially in cases with failed bile duct clearance after sphincterotomy. If endoscopic drainage fails, rapid surgery is mandatory to achieve biliary drainage.
    - iii. Pancreatitis occurs in 1-4% of cases.

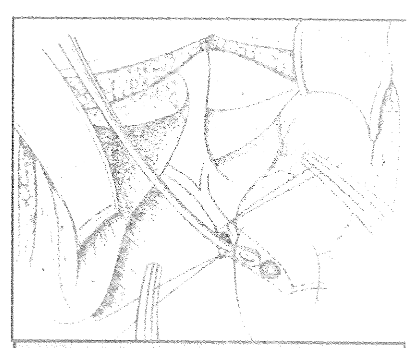


Fig. 32.21. Operative removal of CBD stones.

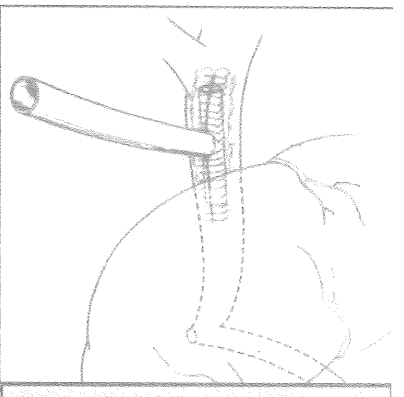


Fig. 32.22. T-tube is placed in CBD after removal of stones..

- **If ERCP is not available or if it fails to clear CBD of stones**, treatment is by operative exploration of CBD and removal of the stones together with cholecystectomy. The operation is called cholecystectomy and choledocholithotomy.
  - Procedure:
    - i. The supraduodenal part of CBD is exposed, opened between 2 stay sutures and all calculi are extracted (Fig. 32.21).
    - ii. A special dilator, (Bake's) is passed through the ampulla to check that there is no stenosis.
    - iii. Flushing of the duct with saline is performed.
    - iv. Some surgeons insert a choledochoscope to check that there are no retained calculi.
    - v. A T-tube is inserted in the CBD (Fig. 32.22), which is closed around it. The long limb of the tube is brought outside the patient. The aim of this tube is to help in the drainage of bile in the early post-operative period.
    - vi. A T-tube cholangiogram can be performed after closure of CBD to check absence of filling defects (completion T-tube cholangiography).
    - vii. Cholecystectomy is then performed.

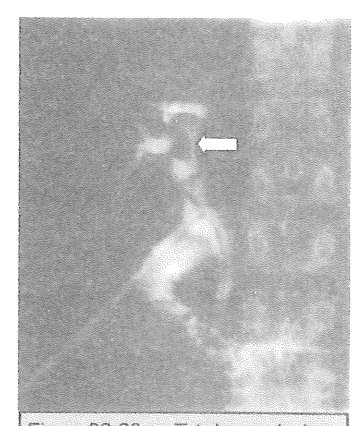


Fig. 32.23. T-tube cholangiogram showing a residual stone in upper part of CBD.

viii. Ten days after surgery a cholangiogram through the T-tube is performed (Fig. 32.23) and if there are no residual stones and there is free flow of the contrast to the duodenum, the tube is removed.

- **Additional procedures to prevent future obstruction**
  - **Indications**
    i. Residual inaccessible stones, e.g., in intrahepatic bile ducts.
    ii. Impacted stone in lower end of CaD.
    iii. Stricture of CBD.
  - **Choice**
    i. Choledocho-duodenostomy if CBD is more than 2cm in diameter.
    ii. Trans-duodenal sphincteriolasty if it is less than 2 cm. This is rarely done nowawdays as it can be performed by endoscopic sphincterotomy.

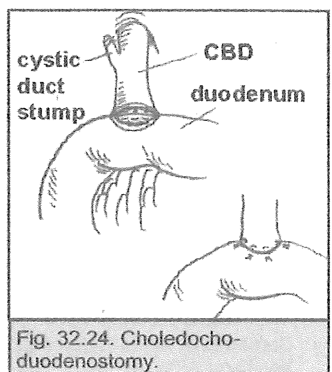
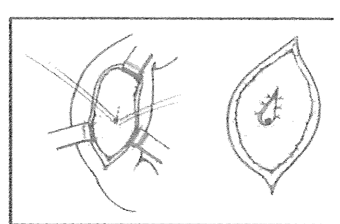


Fig. 32.24. Choledocho-duodenostomy.


Fig. 32.25. Trans-duodenal sphincteroplasty.

Strictures of bile ducts

Congenital biliary atresia (described eailier in this chapter)

Traumatic strictures

Aetiology

This usually follows cholecystectomy or less commonly choledocholithotomy. It may be due to

1. Complete ligation of the common hepatic or common bile duct.
2. Narrowing of the duct due to its inclusion in a ligature.
3. Devitalization of the duct due to rough dissection.
4. Ischaemia of the duct.

The commonest cause of CBD stricture is iatrogenic injury at cholecystectomy.

Diagnosis

Injuries of bile ducts present by either biliary fistula or jaundice following cholecystectomy. ERCP and PTC will localize the site of the stricture which is usually at the common hepatic duct.

Treatment

Operative correction of this stricture is the only treatment but is technically difficult. The remaining stump of the common hepatic duct, if present, or the left hepatic duct is anastomosed to a Roux-en-Y loop of jejunum.

Sclerosing cholangitis

Pathology

This is a condition of unknown aetiology in which the intra-and extrahepatic ducts are involved by multiple strictures separated by normal or dilated segments.

Clinical features

The patient presents by obstructive jaundice and if the condition is untreated, it will eventually lead to liver failure.

Treatment

Anastomosis of a dilated segment of the bile duct to a loop of jejunum. In some patients there is no available dilated segment suitable for anastomosis, these patients are candidates for liver transplantation.

Neoplastic stricture (carcinoma of bile ducts)

Risk factors

Cholangiocarcinoma is more common in elderly males. Predisposing factors include calcular disease, ulcerative colitis, primary sclerosing cholangitis, choledochal cyst and parasitic infestation by clonorchiasis.

Clinical features

Malignant strictures manifest by obstructive jaundice in 90% of patients. Less often the patient complains of pain, cholangitis and anorexia.

Examination reveals hepatomegaly. If the tumour is below the insertion of the cystic duct into the common bile duct, the gall bladder will be distended. If the cholangio-carcinoma is at the porta hepatis (Klatskin's tumour), the gall bladder will be collapsed (an exception to Courvoisier's law).

Investigations

- Abdominal sonar will reveal intrahepatic biliary dilatation. The common hepatic or bile duct will be dilated down to the site of the lesion.
- Percutaneous transhepatic cholangiography (PTC) will demonstrate dilatation of the intrahepatic biliary radicles. It will reveal the proximal dilated portion of the common hepatic duct in high lesions.

Treatment

- Operable lesions at the lower end of CBD are treated by pancreatico-duodenectomy Whipple's operation). Inoperable cases are treated by cholecystojejunostomy (Chapter 33, Fig. 33.17).
- Lesions at the hilum of the liver are rarely operable. Treatment is by anastomosis of any dilated biliary segment above the lesion to a loop of jejunum, or if this is not possible by some form of biliary drainage as percutaneous transhepatic biliary drainage or endoscopic stenting.

Carcinoma of the gallbladder

This lesion is more common in elderly females.

Pathology

- In over 90% of cases gall stones are present.
- Microscopic picture is that of squamous cell carcinoma, adenocarcinoma or a mixture of the two.
- Spread is by direct invasion of the liver and porta hepatis, by lymphatics to the hilar lymph nodes or venous spread to the liver.

Clinical features

1. The tumour is usually diagnosed after pathological examination of the gall bladder that was removed because of chronic calcular cholecystitis.
2. Some patients present by acute cholecystitis as the tumour obstructs the cystic duct.
3. Obstructive jaundice due to obstruction of the bile duct by the tumour or by metastatic lymph nodes in the porta hepatis.
4. Mass in the right hypochondrium.

Treatment

- If the diagnosis is suspected during surgery, the treatment is radical cholecystectomy. This operation includes
    - Excision of the gall bladder.
    - Excision of a wedge of the underlying liver.
    - Clearance of lymph nodes overlying the bile duct.
- Usually the lesion is fairly advanced and inoperable. If there is jaundice, internal stenting may relieve it. The prognosis for this lesion is very poor.

Jaundice

Definition

Jaundice is yellowish colouration of the body tissues and fluids (except the brain, CSF, tears, saliva and milk) which results from accumulation of bilirubin in blood. It becomes manifest when serum bilirubin exceeds 2.5 mg/dL.

Bilirubin metabolism (Fig. 32.26)

1. **RBCs destruction.** Old red blood corpuscles are phagocytosed by the reticuloendothelial cells all over the body but particularly in the bone marrow and the spleen.
2. **Breakdown of haemoglobin.** The liberated haemoglobin undergoes breakdown into globin, and heme.
3. **Biliverdin and bilirubin formation.** Globin enters into the amino acid pool while heme is transformed into biliverdin which is reduced to bilirubin. The latter, being water insoluble, is transported in plsma bound to albumin and constitutes unconjugated bilirubin.
4. **Bilirubin conjugation.** In the liver bilirubin is conjugated with glucuronic acid to form bilirubin diglucuronide (conjugated bilirubin), which is water soluble. Conjugation of bilirubin in the liver is catalyzed by the bilirubin glucuronyl transferase enzyme.
5. **Bilirubin excretion.** Conjugated bilirubin is excreted via the biliary passages to the intestine.
6. **Changes in intestine and reabsorption.** In the terminal ileum and colon, conjugated bilirubin is converted by bacterial enzymes to stercobilinogen which is oxidized to stercolibin responsible for the normal colour of stools. Up to 20% of stercobilinogen is reabsorbed, and 90% of this is promptly reexcreted by the liver, with much of the remainder appearing in urine as urobilinogen.

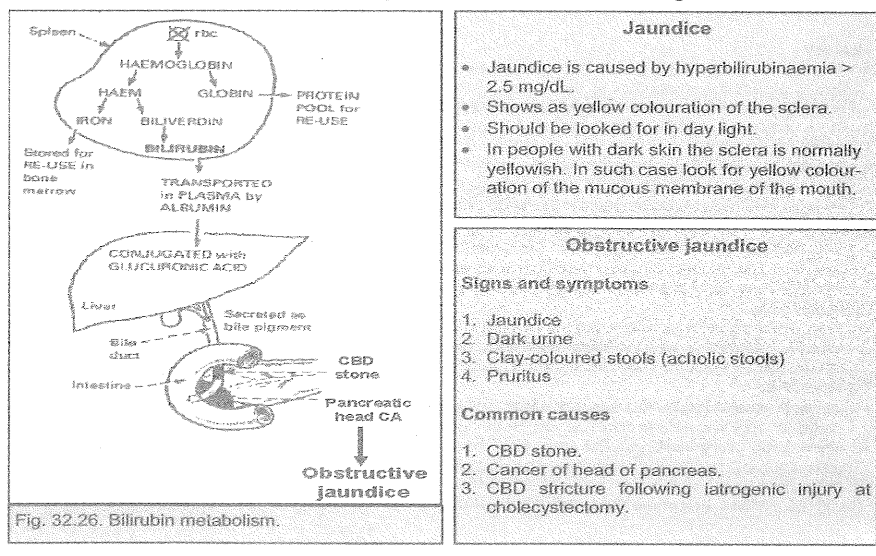


Fig. 32.26. Bilirubin metabolism.

Jaundice

- Jaundice is caused by hyperbilirubinaemia > 2.5 mg/dL.
- Shows as yellow colouration of the sclera.
- Should be looked for in day light.
- In people with dark skin the sclera is normally yellowish. In such case look for yellow colouration of the mucous membrane of the mouth.

Obstructive jaundice

Signs and symptoms

1. Jaundice
2. Dark urine
3. Clay-coloured stools (acholic stools)
4. Pruritus

Common causes

1. CBD stone.
2. Cancer of head of pancreas.
3. CBD stricture following iatrogenic injury at cholecystectomy.

Types and aetiology

According to the aetiology of the excess bilirubin, three types of jaundice are recognized.

1. **Haemolytic** or prehepatic jaundice is due to excessive destruction of red blood corpuscles which occurs in the various types of haemolysis (Chapter 34). Unconjugated bilirubin is elevated. Faecal stercobilinogen and urinary urobilinogen are increased. Bile salts do not accumulate in the serum.
2. **Hepatocellular** or hepatic jaundice is due to liver dysfunction and inability to transform unconjugated bilirubin to the conjugated variety. In addition, due to the presence of intrahepatic cholestasis, some conjugated bilirubin is reabsorbed to the blood. Blood levels of both conjugated and unconjugated bilirubin are elevated. Bile salts levels may also rise. Causes of hepatocellular jaundice include
   a. Viral hepatitis.
   b. Decompensated liver cirrhosis. Bacterial infections as septicaemia and pyaemia.
   c. Drugs. A growing number of drugs including phenothiazine compounds, certain diuretics, testosterone derivatives, oral contraceptives, oral antidiabetic agents, halothane and arsenicals, are known to injure the cellular excretion mechanism and cause intrahepatic cholestasis. A sensitization reaction to the drug is usually responsible.
3. **Obstructive** or posthepatic jaundice is due to obstruction somewhere in the biliary pathway. Conjugated bilirubin will be reabsorbed to the blood and will be excreted in urine. Bile salts will accumulate in the blood. Faecal stercobilinogen and urinary urobilinogen levels are low. Causes of obstructive jaundice include:
   a. Causes in the lumen of the bile ducts, e.g. calculi or parasites as ascaris or fasciola.
   b. Causes in the wall of the bile ducts which include:
      i. Congenital biliary atresia.
      ii. Inflammatory stricture as with sclerosing cholangitis or secondary to an impacted stone which has been present for a long time.
      iii. Traumatic stricture is usually iatrogenic following cholecystectomy, choledocholithotomy or instrumental procedures on the common bile duct.
      iv. Malignant stricture.
   c. Causes outside the bile ducts, e.g., carcinoma of the head of pancreas obstructing the lower part of the common bile duct or a mass of metastatic lymph nodes at the porta hepatis obstructing the hepatic ducts.

Clinical features

History

- The age and sex of the patient may give a hint to the diagnosis. Jaundice in the neonatal period may be due to physiological jaundice of the newborn, Rh incompatibility, neonatal hepatitis, congenital syphilis or congenital biliary atresia. In children haemolytic anaemias should be suspected. Calcular jaundice is more common in middle aged females while malignant obstructive jaundice is commonly seen in elderly males.
- History of drug intake, recent injections and blood transfusion.
- Colour of urine and stools. In haemolytic jaundice urine colour is normal while stools are dark. In obstructive and hepatocellular jaundice, urine is dark while stools are pale.
- Pruritus is absent in haemolytic jaundice.

- Recurrent attacks of haemolytic crisis suggest haemolytic jaundice.
- Depth of jaundice. Haemolytic jaundice is usually of a mild degree while hepatocellular and obstructive jaundice may be of a severe degree. Calcular jaundice is usually fluctuant while malignant jaundice is progressive.
- Pain. In haemolytic jaundice abdominal pain may be present during a haemolytic crisis. Calcular obstructive jaundice is usually accompanied by upper abdominal pain. Malignant obstructive jaundice is usually painless but may be accompanied by dull aching epigastric pain referred to the back.

Examination

- General examination focuses on pallor, pyrexia, the degree of jaundice, Virchow's lymph nodes, cachexia, stigmata of liver insufficiency, bleeding tendency, and oedema of the lower limbs.
- Abdominal examination. Patients with haemolytic anaemia usually have hepatomegaly and splenomegaly. In calcular obstructive jaundice, abdominal examination is usually negative. In malignant obstructive jaundice the liver and gall bladder are commonly palpable. Ascites may be present in advanced cirrhosis or in metastatic malignant lesions.

Investigations

Laboratory

1. Blood picture. In haemolytic jaundice there is anaemia. Special tests for various haemolytic anaemias may be required.
2. Serum bilirubin. Jaundice is easily recognizable when the conjugated bilirubin concentration in the serum reaches 2-3 mg/100 ml or the unconjugated bilirubin level is 3-4 mg/100 ml. In haemolytic jaundice, the uncorugated bilirubin level is increased, in obstructive jaundice the conjugated bilirubin is high, while in hepatocellular jaundice both types of bilirubin are increased.
3. SGOT (AST) and SGPT (ALT). In haemolytic jaundice the level of transaminases will be normal. In acute hepatitis the level will be very high while in cirrhosis there will be moderate increase. In the early stages of obstructive jaundice the level will be normal but with prolonged obstruction, some degree of parenchymatous damage occurs and the level of these enzymes will increase.
4. Alkaline phosphatase. In haemolytic jaundice the level of alkaline phosphatase is normal, in hepatocellular jaundice, there may be slight increase, while in obstructive jaundice there is moderate increase in the level of this enzyme particularly in malignant obstruction.
5. Prothrombin time and concentration are normal in haemolytic jaundice. There is pmlongation of prothrombin time and diminished prothrombin concentration in both hepatocellular and obstructive jaundice. To differentiate both conditions, a course of IV vitamin K is prescribed for a few days. In obstructive jaundice the prothrombin parameters will improve but not in hepatocellular jaundice.
6. Serum albumin is low in patients with cirrhosis or after prolonged malignant cachexia.
7. Faecal stercobilinogen is high in haemolytic jaundice but is low in hepatocellular and obstructive jaundice.

Imaging

1. **Abdominal ultrasound**. This is undoubtedly the first investigation to be ordered. It will demonstrate dilatation of intrahepatic biliary radicles in patients with obstructive jaundice. Further data provided by ultrasound have been discussed before.

2. **CT scan** is particularly indicated in patients suspected of having abdominal malignancy, e.g., carcinoma of the head of pancreas. It will clearly demonstrate the site and extent of the tumour, invasion of adjacent structures, metastatic lymph nodes or liver deposits.
3. **ERCP** is particularly indicated in patients with obstructive jaundice suspected of having a lesion involving the lower end of the common bile duct, e.g., ampullary carcinoma.
4. **PTC** is particularly advised in patients with obstructive jaundice suspected of a having a lesion involving the upper end of the common bile duct, e.g. postoperative stricture or carcinoma of the hepatic ducts.

Cholecystectomy

Removal of the gall bladder is an operation which needs wide knowledge of the anatomy and congenital anomalies of the biliary tree. It requires meticulous dissection. If performed by a competent surgeon, it has a low morbidity and mortality, even in difficult cases.

Fig. 32.27. Open cholecystectomy.

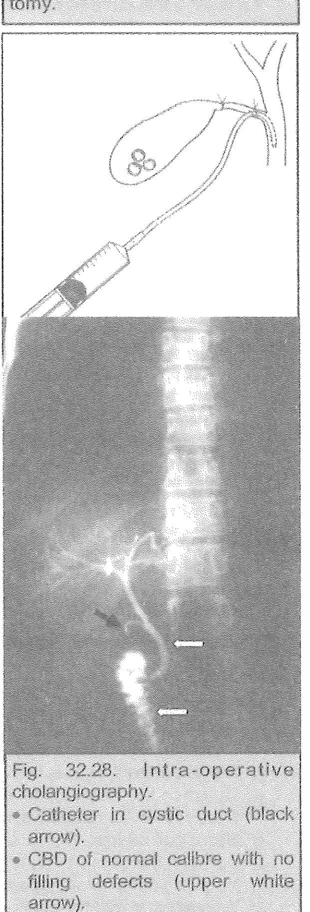


Fig. 32.28. Intra-operative cholangiography.
- Catheter in cystic duct (black arrow).
- CBD of normal calibre with no filling defects (upper white arrow).
- Free flow to duodenum (lower .

Indications

1. **Gall stones**
   a. Stones that cause biliary colic.
   b. Acute calcular cholecystitis. The operation may be done early in the course of the attack, i.e., in the first three days, or late, i.e., after 6 weeks of subsidence of the attack by conservative treatment.
   c. Common bile duct stones. Cholecystectomy is done because the gall bladder is the usual source of CBD stones. Its removal aims at prevention of recurrence.
   d. Silent gall stones (mentioned previously).
2. Acute non-calcular cholecystitis.
3. Carcinoma of gall bladder.

Conventional (open) cholecystectomy

1. **Anaesthesia**. General.
2. **Incision**. A right subcostal or paramedian incision is performed. A paramedian incision can be extended and it allows full exploration. It is particularly indicated if the subcostal angle is narrow. A subcostal incision gives direct access to the gall bladder. It is a muscle cutting incision and may injure the ninth or tenth intercostal nerve.
3. **Exposure**. After exploration, one assistant pulls the liver upwards while the other assistant pulls the duodenum downwards thus exposing the free margin of lesser omentum.
4. **Displaying the Y junction**. Dissection starts by incising the petitoneum on the free margin of lesser omentum to expose the cystic duct, common hepatic and common bile ducts.

5. **Cystic artery control**. The cystic artery is usually at a higher and more posterior plane than the cystic duct. It is ligated and divided (Fig. 32.27).
6. **Intra-operative cholangiography** (IOC) (Fig. 32.28). An operative cholangiogram can be performed at this step. A ligature is applied to the cystic duct on the side of the gall bladder, a tiny opening is made in the cystic duct, a fine cannula introduced and a ligature is applied over it. About 4 ml of dilute biligrafin are injected and two films are taken. The value is to demonstrate any stone in the common bile dud which appears as a filling defect with failure of passage of the dye to the duodenum.
7. **Cystic duct division**. The cystic duct is ligated about 5 mm lateral to the common bile duct and is then divided.
8. **Gall bladder removal and haemostasis**. The gall bladder is dissected from its bed. A few bleeding points may be present in the gall bladder bed and are controlled by diathermy.
9. **Closure**. Usually a drain is placed in the hepato-renal pouch. The drain is left for a few days. The abdominal wall is closed in layers.

Laparoscopic cholecystectomy (LC)

This procedure has been one of the major advances in the surgical field in the 1990s.

Technique

The idea of the procedure is to induce pneumoperitoneum using CO_2 gas. Then through 4 small ports (Fig. 32.29), a telescope and instruments are introduced in the peritoneal cavity. The telescope is connected to a video camera that allows the operative field to be projected on a monitor. By using special graspers and instruments, the surgeon can perform cholecystectomy following the same steps as in open surgery (Fig. 32.30).

If during the operation difficulties or complications arise, e.g., uncontrollable bleeding, the operation is converted into open surgery to gain better control on the situation. The patients safety comes first.

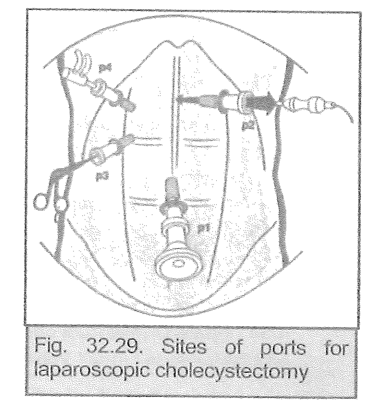


Fig. 32.29. Sites of ports for laparoscopic cholecystectomy

Advantages

1. Post-operative pain is less severe, as there are only 4 small wounds.
2. Post-operative stay in the hospital is short, i.e., 1-2 days.
3. Post-operative convalescence is more smooth and rapid allowing early return to work.
4. Better cosmetic result.

Contraindications

1. Pregnancy. There is no room to induce pneumo-peritoneum and CO_2 gas may be toxic to the foetus.
2. Carcinoma of gall bladder.
3. Bleeding diathesis.
4. Compromise of the cardiovascular or respiratory functions. It is to be noted that CO_2 gas is used to induce the pneumoperitoneum and during the whole procedure the intra-abdominal pressure is very high.

Difficult cases

1. Marked obesity as it is difficult to introduce the ports.

2. Liver cirrhosis. Major bleeding may occur from the bed of the gall bladder.
3. Empyema of the gall bladder as it is difficult to hold the tense gall bladder and there might be adhesions around it.
4. Previous upper abdominal surgery is a relative contraindication. There may be many adhesions between the viscera and the abdominal wall.

Complications of cholecystectomy

Complication common to all operations, e.g., atelectasis, DVT, wound infection, or incisional hernia.

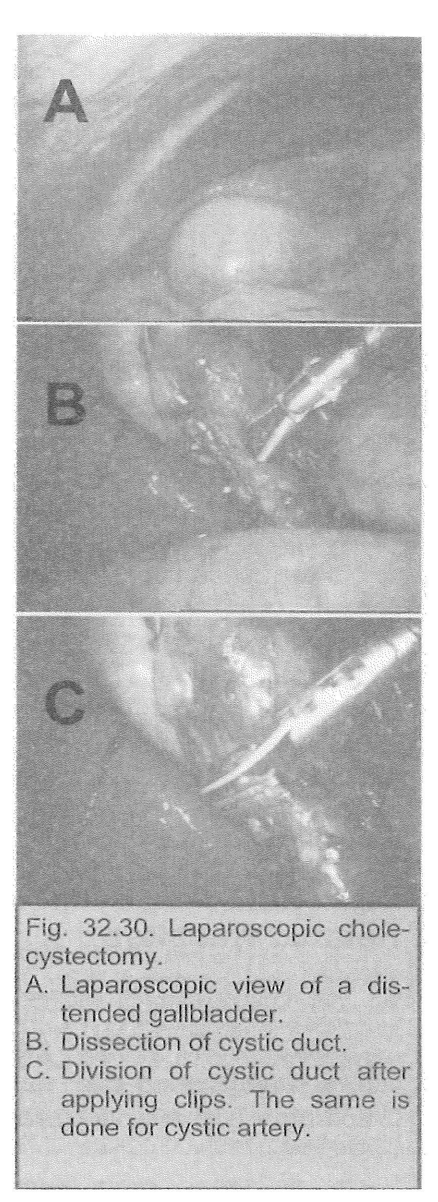


Fig. 32.30. Laparoscopic cholecystectomy.
A. Laparoscopic view of a distended gallbladder.
B. Dissection of cystic duct.
C. Division of cystic duct after applying clips. The same is done for cystic artery.

Complications which are specific to cholecystectomy

1. **Haemorrhage** may be primary or reactionary. Primary haemorrhage may be due to injury of the cystic artery, right hepatic artery or bleeding from the gall bladder fossa. If bleeding occurs a gauze pack is applied for few minutes, after which the bleeding point will be visualized and ligated. If the bleeding is serious Pringle's technique (compression of the free margin of lesser omentum between the index and thumb for few minutes) will allow visualization of the bleeding point. If reactionary haemorrhage affects the haemodynamic status of the patient, exploration is mandatory.
2. **Biliary leakage** This may present as excess bile leakage via the drain or as biliary peritonitis. It is due either to slipping of the ligature on the cystic duct or to injury of the biliary apparatus. ERCP and stenting of the CBD for few days may solve the problem.
3. Subphrenic collection of bile, or blood or subphrenic abscess. Ultrasound examination will confirm the diagnosis.
4. **Jaundice**. This is a serious problem. Rarely, it is due to a medical cause, e.g., halothane anaesthesia. Post-cholecystectomy jaundice, however, is usually due to a missed stone in the CBD or to injury of the common bile or hepatic duct. ERCP will diagnose the aetiology.
5. **Post-cholecystectomy syndrome**. This term is used when the patient still complains of pain or dyspepsia following surgery. It is due to
   a. Non biliary causes The original diagnosis might be wrong, and the patient may have another cause for the symptoms, e.g., peptic ulcer, hiatus hernia, chronic colitis, or irritable bowel.
   b. Biliary dyskinesia This is a vague problem in which there is some sort of neuromuscular incoordination of the biliary apparatus that results in failure of relaxation of the sphincter of Oddi.
   c. Problems related to the operation as missed stone in the common duct, operative injury to the common hepatic or bile duct causing partial stricture, or a missed stone in the cystic duct stump. ERCP can demonstrate these problems.

PANCREAS

Surgical anatomy

General features

The pancreas is an upper abdominal retroperitoneal organ that has the shape of a pistol. The handle of the pistol lies in the duodenal C-loop, and the barrel extends horizontally to the left towards the hilum of the spleen.

CHAPTER CONTENTS

Parts

The organ is formed of the following parts: (Fig. 33.1)

- The head is housed in the duodenal "C'. The common bile duct is posterior to the head and is partially embedded within it. It also lies infront of the inferior vena cava. As the superior mesenteric vessels emerge at the lower border of the pancreas they lie infront of a tongue like projection called the uncinate process.
- The neck is the part of the pancreas that lies infront of the junction of the superior mesenteric and splenic veins to form the portal vein.
- The **body** is related posteriorly to the aorta and the splenic vein, and anteriorly to the lesser sac (omental bursa) which separates it from the posterior surface of the stomach. The splenic artery runs to the left just above the body.
- The tail is related to the splenic hilum, and the splenic vessels.

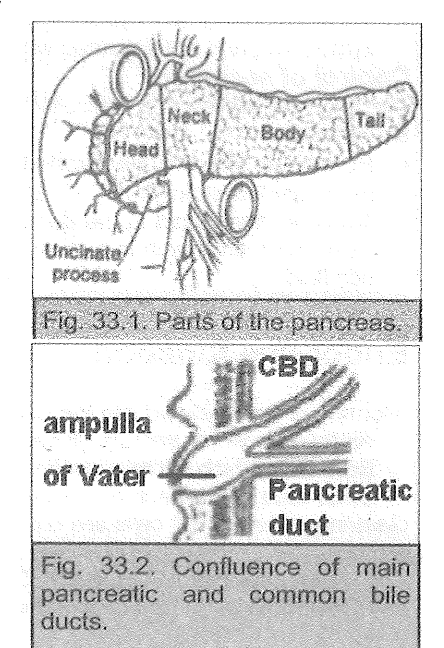


Fig. 33.1. Parts of the pancreas.

Fig. 33.2. Confluence of main pancreatic and common bile ducts.

Ducts

1. **The main pancreatic duct** (the duct of Wirsung) joins the common bile duct at the ampulla of Vater, which opens in the middle of the medial surface of the second part of the duodenum on the major duodenal papilla (Fig. 33.2). The common opening is surrounded by the sphincter of Oddi.
2. The accessory duct (duct of Santorini) may join the main duct or may open separately above it on the minor duodenal papilla.

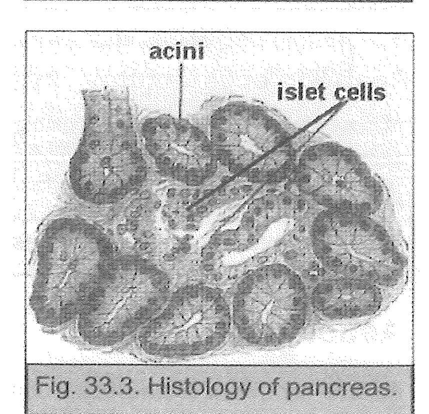


Fig. 33.3. Histology of pancreas.

Blood supply

Arteries

- Branches from the **splenic** artery supply the body and tail.
- The superior and inferior pancreaticoduodenal arteries form an arcade that lies in the concavity of the duodenal "C" and supplies it and the head of the pancreas.

Veins

Venous blood drains to corresponding veins, and ultimately to the portal vein.

Microscopic anatomy

The pancreas has a thin capsule that sends in septa dividing it into lobules. It shows two main features (Fig. 33.3).

- Acini formed of low columnar cells that secrete digestive enzymes. These are collected by a network of small ducts that are lined by secretory cells.
- Islets of Langerhans which are aggregations of endocrine cells that are concentrated in the left half of the gland.

Surgical physiology

The pancreas is a mixed exocrine and endocrine organ.

Exocrine function

Amount

The acinar cells of the pancreas secrete 1-2 litres of alkaline enzyme-rich juice daily.

Enzyme content

- Lipase.
- Amylase.
- Proteolytic enzymes trypsinogen and chymotrypsinogen. These two are secreted in an inactive proenzyme form, and are activated when they reach the intestine.

Control of secretion

The exocrine secretion is controlled by the gut hormones and by the autonomic nervous system.

Stimulation

- **Hormones**. Food in the duodenum and proximal jejunum stimulates their mucosa to secrete the hormone cholecystokinin (CCK), which in tum stimulates the release of enzymes from the acinar cells. On the other hand, acid in the duodenum and proximal jejunum stimulates the mucosa to secrete the hormone secretin, which in turn stimulates the ductal epithelium to secrete an alkaline watery bicarbonate- rich fluid.
- **Vagus**. The release of hormone is also stimulated by parasympathetic vagal discharge.

Inhibition

- Sympathetic stimulation
- GIT hormones; somatostatin and glucagon.

Endocrine function

Hormones are released into the blood stream from the islets of Langerhans that contain the following cells.

Alpha-cells which secrete glucagon.

Beta-cells which secrete insulin.

Delta-cells which secrete somatostatin.

PP cells secrete pancreatic polypeptide.

Gastrin-producing "G" cells are not found in a normal pancreas but appear in the rare Zollinger-Ellison syndrome.

The above cells belong to the APUD series, and have, in common, the capacity of Amine Precursor Uptake and Decarboxylation (removal of the carboxyl end of the molecule).

Congenital Anomalies:

Annular pancreas:

This consists of a ring of pancreatic tissue that surrounds the second part of the duodenum. It may cause duodenal obstruction, with half of the cases presenting during the first year of life. Vomiting is the main symptom and jaundice may be present. The diagnosis is reached by contrast radiological studies. Treatment is by duodeno-jejunostomy.

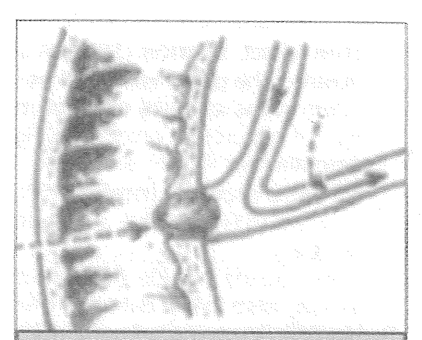


Fig. 33.4. Acute pancreatitis is one of the complications of common bile duct (CBD) stones.

Classification of pancreatitis

- Acute pancreatitis after which the gland returns to anatomical functional normality.
- Chronic pancreatitis is associated with permanent derangement of structure and function.

Some patients suffer from relapsing acute pancreatitis, enjoying relatively normal health between attacks.



Acute pancreatitis

Acute inflammation of the pancreas is one of the causes of acute abdominal pain. It is a serious condition that leads to death in 10% of cases.

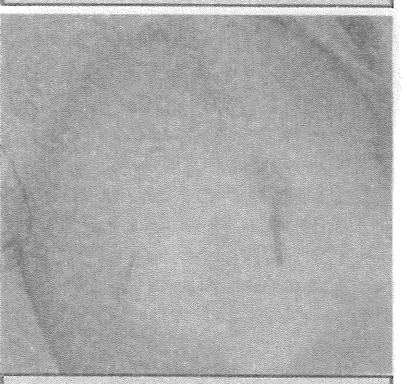


Fig. 33.5. Cullen's and Grey Turner's signs.

Causes of acute upper abdominal pain.

1. Acute cholecystitis (and biliary colic).
2. Perforated peptic ulcer.
3. Acute pancreatitis.
4. Acute mesenteric vascular occlusion.
5. Leaking aortic aneurysm.
6. Acute myocardial infarction (basal).

All are serious diseases.

Aetiology

Several conditions are associated with acute pancreatitis.

1. **Bile duct stones (50%).** A stone lodged in the extreme lower part of the common bile duct is likely to obstruct the pancreatic duct as well-One of the theories suggested to explain acute pancreatitis is that a stone in the ampulla of Vater may lead to regurgitation of bile into the pancreatic duct leading to activation of pancreatic enzymes (Fig. 33.4). It is now realized that the mere passage of a stone through the ampulla may initiate an attack.
2. **Excess alcohol intake** (35%).
3. **Trauma** may be accidental, operative, or with endoscopic retrograde cholangio-pancreatography (ERCP). The latter is the third most common cause of pancreatitis. .
4. Rare causes include viral infection (mumps), hyperparathyroidism, corticosteroids and hypertriglycerinaemia.
5. No definite cause is found in 20% of cases. These are referred to as idiopathic pancreatitis, however, it has been suggested that most of these cases are secondary to the passage of minute stones thorough the sphincter of Oddi.

Pathology

Pathogenesis

In acute pancreatitis there is premature activation of the pancreatic digestive enzymes while still in the pancreas. This leads to autodigestion of the gland. The exact mechanism of enzyme activation is not clearly understood. Inflammatory cytokines are produced and trigger an inflammatory cascade which leads to the systemic inflammatory response syndrome (SIRS).

Morbid physiology

- The condition is likened to a major burn pf the peritoneum with abundant protein-rich exudate.
- The enzyme lipase which is released from the pancreas splits fat in the omentum and peritoneum producing glycerol and fatty acids. These acids bind with calcium forming insoluble calcium soap which manifests as white spots of fat necrosis in the mentioned areas.
- The binding with calcium is likely to produce hypocalcaemia in severe cases.
- The proteolytic activity of the liberated pancreatic enzymes produces vasoactive kinins and other chemical mediators which are greatly responsible for the haemodynamic and respiratory consequences,

Gross appearance

- The severity of pancreatic inflammation ranges from mild oedema, to haemorrhage (haemorrhagic pancreatitis), and severe necrosis (necrotizing pancreatitis).
- Ascites, which may be haemorrhagic.
- White spots of fat in the peritoneunm.

Complications

Acute pancreatitis is a serious disease that is known for its many complications.

Systemic complications in the form of multiple system failure, to a large extent simulating those of a major burn (refer to SIRS in chapter 6). All systems of the body are likely to be affected. The occurrence of any of these complications worsens the prognosis, and if improperly treated, one system failure leads to another, and the chain ends in death.

1. Shock from loss of plasma into the peritoneum and into the retroperitoneal space, and from the loss of blood in haemorrhagic cases.
2. Adult respiratory distress syndrome (ARDS is discussed in Chapter 28) with consequent respiratory failure in severe cases.
3. Renal failure follows prolonged hypovolaemia.
4. Consumption coagulopathy (coagulation system failure).
5. Paralytic ileus and acute gastroduodenal stress ulceration and haemorrhage.
6. Tetany sometimes occurs because of hypocalcaemia.

Local complications

1. Pancreatic pseudocyst (discussed later).
2. Pancreatic abscess (4.5%).

Clinical features

Symptoms

1. Upper abdominal pain is the main symptom and is present in all cases. Pain has an acute onset and gradually intensifies in severity. Frequently pain becomes agonizing. It commonly radiates to the back. The pain may follow a heavy meal or ingestion of alcohol.
2. Vomiting and reaching are often present.

Signs

- General examination
    - Fever and tachycardia are common.
    - Hypovolaemic shock in severe cases.
    - A tinge of jaundice is sometimes observed.
    - The patient commonly sits leaning forward or draws up the knee as these positions partially relieve the pain.
- **Local signs** are less impressive than expected from the severity of pain.
    - There are only mild tenderness and rigidity. This is explained by the position of the pancreas being a retroperitoneal organ lying far from the sensitive parietal peritoneum.
    - Bruising around the umbilicus (Cullen's sign), and in the loin (Grey Turner's sign) are rare late manifestations that result from slow trickling of the blood tinged exudate from the retroperitoneal space (Fig. 33.5).
    - Two to three weeks after the acute episode, an upper abdominal swelling is sometimes observed (pancreatic pseudocyst).

Differential diagnosis

Acute pancreatitis should be differentiated from other causes of acute upper abdominal pain.

1. Perforated peptic ulcer.
2. Acute cholecystitis and biliary colic.
3. Acute mesenteric vascular occlusion.
4. Leaking aortic aneurysm.
5. Acute myocardial infarction.

All these causes of acute upper abdominal pain are serious and, if neglected, may be fatal.

Investigations

The diagnosis of acute pancreatitis is mainly accomplished by exclusion of other causes of acute abdominal pain.

1. **Serum amylase** is usually elevated within a few hours to levels higher than 1000 IU/dl (normal value is 100-300 IU/dL). It remains elevated for 2-3 days. The problem with serum amylase is that it is not specific for acute pancreatitis. It is elevated in other conditions as perforated peptic ulcer, acute cholecystitis, intestinal obstruction and acute mesenteric ischaemia. The enzyme level in these cases, however, usually does not exceed 500 IU/dL.
2. **Urinary amylase**. As the serum amylase is cleared out by the kidney, urinary amylase level can be used for diagnosis of patients who present after two days of the start of pain.
3. **Serum lipase**. Lipase has a slightly longer half-life. It is particularly useful in cases of delayed presentation. Elevated lipase levels are more specific to the pancreas than amylase levels.
4. **Arterial blood gases** to detect cases who will need mechanical ventilation.
5. The following biochemical changes are commonly found; mild bilirubin elevation, hypocalcaemia, hypoproteinaemia, elevated blood urea, and hyperglycaemia.
6. The blood picture commonly shows leucocytosis. The haematocrit is usually elevated because of fluid loss, but is lowered in cases of haemorrhagic pancreatitis.

7. **Plain X-ray** of the abdomen usually shows a dilated short segment of the small intestine that is commonly referred to as a "sentinel loop". There may also be distension of the transverse colon and collapse of the descending colon (colon cut-off sign).
8. **Abdominal ultrasound** may show gall bladder stones. Stones that are impacted in the lower end of the common bile duct are usually not seen, yet their presence is indicated by dilatation of the biliary tree.
9. **CT scan with IV contrast** is very helpful in the diagnosis. It reveals enlargement of the pancreas, peripancreatic oedema and intraperitoneal fluid. Pancreatic necrosis is diagnosed when a big part of the parenchyma is not enhanced after contrast injection (Fig. 33.6). The presence of necrosis is a strong indicator of severity of the attack. A CT scan should be performed at least 48 hours after the onset of symptoms in order to detect the extent of necrosis.



Fig. (33.6): CT scan showing necrotizing pancreatitis

10. **Magnetic resonance cholangiopancreatography** (MRCP) may be done if choledocholithiasis is suspected.
11. **ECG**, and the cardiac enzyme creatine phosphokinase (CPK) are done to exclude myocardial infarction.

Assessment of severity:

Identifying patients in greatest need of aggressive treatment by differentiating their disease severity as mild or severe is needed to pick up patients who will require intensive therapy.

Ranson's criteria as shown below, are used to identify patients at higher risk of mortality:

On admission		**Initial 48 hours**	
Age	>55 years	Haematocrit decrease	>10%
White cell count	>16000/uL	BUN elevation	>5 mg/dl
Blood glucose	>200 mg/dl	Serum calcium	<8 mg/dl
Serum LDH	>350 IU/L	Arterial PO_2	<60 mm Hg
AST	>250 IU/L	Base deficit	>4 mEq/L

Treatment

The treatment of acute pancreatitis is essentially conservative. Hospitalization is necessary. Severe cases re admitted to the intensive care unit (ICU).

Conservative treatment

The treatment of acute pancreatitis is essentially conservative. The treatment mainly aims at support of the different body systems, and is mainly remembered as the 'R' regimen.

1. Relief of pain by pethidine (meperidine). This is combined with an atropine derivative to counteract the spasm of the sphincter of Oddi induced by opiates.
2. Replacement of the lost fluids by crystalloids, and plasma. Blood is used in hemorrhagic cases. Replacement is monitored by vital signs, urine output, central venous pressure (CVP), and haematocrit. Calcium is added to the infusions as required.
3. Rest of the pancreas and bowel by keeping the patient NPO (no oral intake), and by nasogastric suction. As the patient's anorexia and pain resolve, feeding is introduced enterally, starting with a low fat diet. For patients with severe attacks total parenteral nutrition is induced.
4. Respiratory support by oxygen mask, or by mechanical ventilation in cases of respiratory failure.
5. Resistance of infection by prophylactic antibiotics. The value of antibiotics is controversial, yet it may reduce the incidence of infection in cases of necrotizing pancreatitis. The preferred antibiotics are those of the carbapenems class, e.g. imipenem/cilastatin or meropenem.
6. Removal of an obstructing bile duct stone by endoscopic retrograde cholangiopancreatography (ERCP) and sphincterotomy in the acute attack is recommended if there is obstructive jaundice or cholangitis. Otherwise this intervention is not to be recommended during the attack.
7. Reassessment when the symptoms completely resolve by ERCP. In biliary pancreatitis cholecystectomy and removal of common duct stones, if not endoscopically removed, is performed within the same hospital admission. Alcoholic patients are advised to give up alcohol intake.

Surgical treatment is indicated for:

1. A doubtful diagnosis. Conditions as perforated peptic ulcer, intestinal infarction, and intestinal obstruction are fatal if not urgently treated by surgery, and if these cannot be excluded, it is safer to explore the abdomen. At laparotomy pancreatitis shows as white spots of fat necrosis, mild ascites, and retroperitoneal oedema, haemorrhage, or pancreatic necrosis.
2. Drainage of a pancreatic abscess, or a persistent pseudocyst that does not resolve in six weeks. The abscess is drained externally by a tube, while a cyst is internally drained to the stomach or to a jejunal loop.
3. In severe necrotizing pancreatitis as detected by a CT scan necrotic tissue is excised and two tube drains are left in the peritoneal cavity to allow postoperative lavage.

Pancreatic pseudocyst

Pathology

- **Nature.** This is a collection of pancreatic secretion and inflammatory exudate within a lining of inflammatory tissue rather than epithelium, and that is why it is called a pseudocyst (false cyst).
- **Aetiology**. The pseudocyst develops in 10% of cases of acute pancreatitis. The cyst usually develops 2-3 weeks after the acute attack. The next common cause is pancreatic trauma.
- **Usual site**. The cyst is commonly located in the lesser sac between the pancreas and the stomach (Fig. 33.7).

- Possible complications include infection, haemorrhage, and rupture.

Clinical features

- A small pseudocyst is painless and may be discovered by follow up sonography.
- A large one causes discomfort and manifests clinically as an upper abdominal swelling, that sometimes reaches the size of a melon.

Investigations

- Barium meal examination shows forward displacement of the stomach in a lateral view (Fig. 33.7).
- The most accurate and convenient diagnostic measures are abdominal ultrasound and CT scan (Fig. 33.8).



Fig. 33.7. A pseudocyst usually pushes the stomach forwards. In this case it pushes it laterally, as well, as shown on a P-A view.

Treatment

- Most of these cysts resolve spontaneously, and all that is needed is clinical and ultrasound follow up.
- A persistent pseudocyst that does not resolve in six weeks is surgically drained. Waiting for this period is important to ensure that the cyst is not resolving spontaneously, and to allow the development of a strong cyst wall that can hold sutures. The cyst is internally drained to the stomach or to a jejunal loop. Cystogastrostomy (Fig. 33.9) is done by making an incision in the anterior wall of the stomach, then incising the posterior gastric wall and the cyst wall which are adherent to each other. A continuous interlocking suture binds the posterior gastric wall to the cyst wall securing haemostasis and making an opening between the cyst and the stomach. Postoperatively the cyst gradually disappears.

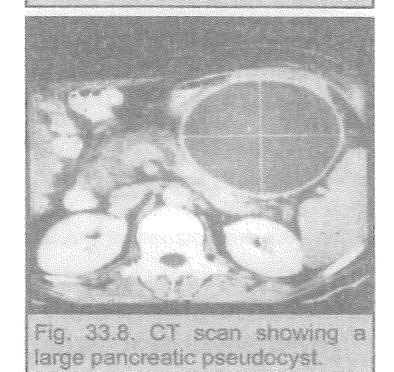


Fig. 33.8. CT scan showing a large pancreatic pseudocyst.

Chronic pancreatitis

Chronic pancreatitis is rare in Egypt.

Aetiology

1. The main causative factor is the over-intake of alcohol.
2. Next common cause is a stone in the lower end of the common bile duct.

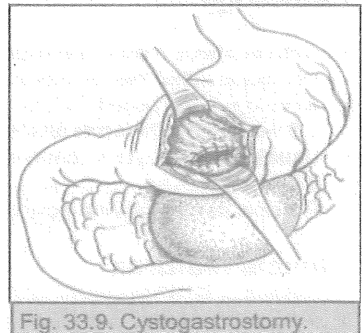


Fig. 33.9. Cystogastrostomy.

Pathology

- There is always a stricture or strictures of the pancreatic duct.
- Irreversible replacement of the acinar cells with fibrous tissue.
- Gradual loss of both exocrine and endocrine functions.
- In some cases inflammation and fibrosis of the head of the pancreas may obstruct the lower part of the common bile duct, leading to obstructive jaundice.

Clinical features

1. Pain is classically in the epigastrium and radiates to the back. It may be mild or severe, and continuous or interrupted. Pain commonly causes drug addiction for analgesics.
2. Malabsorption from loss of enzymes produces steatorrhoea in one third of cases, together with loss of weight, and weakness. Stools typically are soft, greasy, and foul-smelling.
3. Diabetes mellitus develops with extensive damage of the pancreas. It occurs in one third of patients.
4. There are few physical signs. Malnutrition and a tinge of jaundice (in 3% of cases) may be evident. There is epigastric tenderness.

Investigations

Aim of investigations is to define:

1. The degree of exocrine and endocrine functional impairment.
2. The extent of structural damage to the duct system.

Laboratory

There is no laboratory test which is specific for chronic pancreatitis.

1. Steatorrhoea is assessed by measuring faecal fat excretion over 3-5 days while fat intake is controlled at 100 g/day. Normal individuals excrete less than 5 g/day.
2. Lundh test. Secretin and pancreozymin stimulation shows reduced pancreatic secretion.
3. Glucose tolerance test.
4. Serum bilirubin and other liver function tests.

Imaging

- **Plain x-ray** may show calcification or stones.
- **Ultrasound and CT** scan detect pancreatic enlargement and show associated biliary pathology.
- Endoscopic retrograde cholangiopancreatography **(ERCP)** is of great value and should be performed if operation is considered. The architecture of the pancreatic duct is revealed. Common features include strictures, stones, cysts, or alternating strictures and dilatations of the pancreatic duct, which give an appearance of that is known as the "chain of lakes". Pure pancreatic juice can be obtained for cytology to exclude malignancy. ERCP is sometimes of therapeutic value as well. Endoscopic division of the sphincter of Oddi (sphincterotomy) is done in cases of obstruction of the opening of the duct with stone, or with stenosis of the duodenal papilla.
- Magnetic resonance cholangioancreatography **(MRCP)** provides information on the pancreatic ductal system. MRCP is relatively safe, reasonably accurate, noninvasive, fast, and very useful in planning surgical or endoscopic intervention.

Pancreatitis basic facts

- Pancreatitis begins when the digestive enzymes become active inside the pancreas and start "digesting" it.
- Pancreatitis has two forms: acute and chronic.
- Pancreatitis is often caused by gallstones or by alcohol abuse.
- **Acute pancreatitis.**
  - Acute pancreatitis is an emergency.
  - Symptoms of acute pancreatitis include severe acute pain in upper abdomen, nausea, and vomiting. Shock is commonly present with moderate tenderness and rigidity.
  - Acute pancreatitis may be fatal. It should be differentiated from other causes of acute upper abdominal pain, which are equally serious. Serum amylase and CT scan help in diagnosisof pancreatitis. U/S, X-ray and ECG help in exclusion of other causes.
  - Treatment for acute pancreatitis is essentially medical by support of different body systems. It requires hospitalization, better in an ICU. Surgery is seldom needed.
- **Chronic pancreatitis**
  - Acute pancreatitis becomes chronic when pancreatic tissue is permanently destroyed and fibrosis develops.
  - Treatment for chronic pancreatitis includes easing the pain; eating a high-carbohydrate, low-fat diet; and taking enzyme supplements. Surgery is sometimes needed as well (partial pancreatectomy or pancreatico-jejunostomy).

- Endoscopic ultrasonography may be the best test for imaging the pancreas, but requires a highly skilled gastroenterologist. The most predictive endosonographic feature is the presence of stones.

Treatment

Abstinence from alcohol intake is essential.

Conservative treatment is adopted for the majority of cases.

1. Control of pain by non-opiate analgesics, to avoid addiction.
2. Correction of the malabsorption by intake of pancreatic enzyme tablets with meals. A low fat died with oral supplementation of fat soluble vitamins.
3. Control of diabetes by insulin therapy.

Surgery is indicated for persistent uncontrolled pain. Options are:

1. **Resection** of a part of the pancreas is done when it is severely damaged and the rest of the organ is relatively healthy. This is either a distal pancreatectomy excising the body and tail of the pancreas, or a pancreaticoduodenectomy (Whipple operation) if the head of the organ is to be excised. After surgery, pain usually gets better, but diabetes and steatorrhoea still need treatment.
2. Drainage of a dilated pancreatic duct by pancreaticojejunostomy.
3. Thoracoscopic splanchincectomy. Thoracoscopic division of the splanchnic nerves (splanchnicectomy) interrupts the pain pathways from the pancreas and relieves pain of chronic pancreatitis.

Table 33.1. Classification of pancreatic neoplasms.

	Exocrine
Benign	Adenoma
Malignant	Adenocarcinoma
	Endocrine
Alpha cells	Glucagonoma
Beta cells	Insulinoma
Delta cells	Somatostatinoma
Non-Beta cells	Gastrinoma

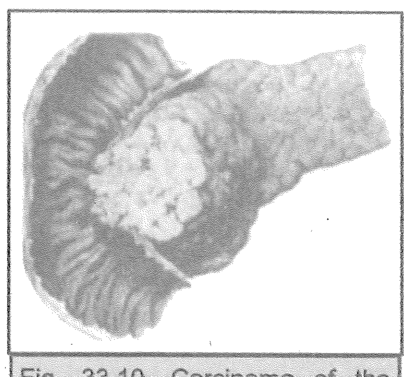


Fig. 33.10. Carcinoma of the head proper.

Pancreatic neoplasms classification

The classification of pancreatic neoplasms is shown in table 33.1.

- Pancreatic neoplasms commonly arise from its exocrine tissues.
- Endocrine neoplasms are rare, and as they arise from APUD cells, they are termed apudomas. They are interesting in regard to their metabolic consequences, and the possibility of associated APUD neoplasms in other organs, forming what is known as multiple endocrine neoplasia (MEN) syndrome (see chapter 29).

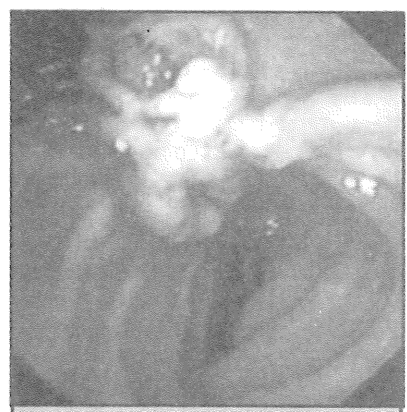


Fig. 33.11. Periampullary carcinoma arising from the duodenal papilla, as shown at endoscopy.

Pancreatic carcinoma

Carcinoma of the pancreas is increasing in incidence. It affects males more than females. The tumour's peak incidence lies between 55 and 70 years of age. The prognosis is extremely poor, the 5 year survival rate is less than 5%.

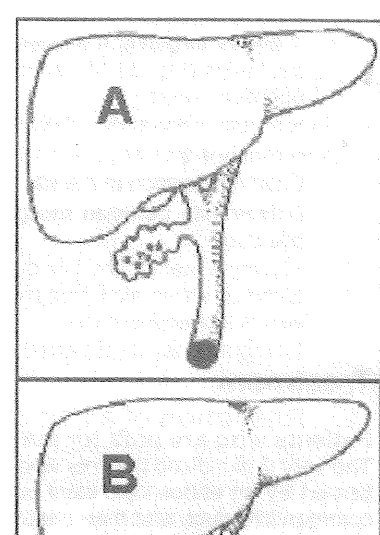
Aetiology

The exact aetiology is unknown. Factors that are thought to increase the risk include smoking, high protein, high fat food, alcohol abuse and chronic pancreatitis.

Pathology

Gross picture

- The growth is infiltrating, hard, and irregular.
- Sixty percent of the lesions are situated in the head, the remainder are in the body and tail.
- Of the head tumours, two thirds are in the head proper (Fig. 33.10), and one third is a periampullary carcinoma. The latter arises either from the ampulla of Vater, the duodenal papilla (Fig. 33.11), or the lower end of the common bile duct.



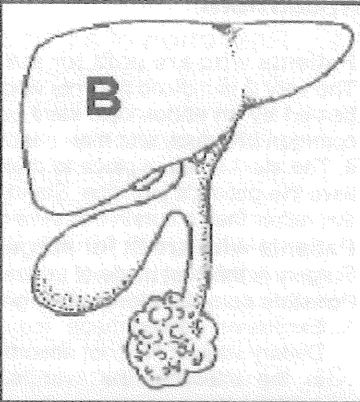


Fig. 33.12. Palpation of gall bladder in obstructive jaundice cases.
A Calcular: usually impalpable.
B Malignant: commonly enlarged and palpable.

Courvoisier's law states that **"If the gall bladder is palpable in the presence of jaundice, the jaundice is unlikely to be due to stone".**
Explanation: The gall bladder fails to distend as it is the seat of fibrosis as a result of chronic calcular cholecystitis.
Courvoisier's law has exceptions.

Microscopic picture

The tumour is an adenocarcinoma arising from the ductal system, with variable degrees of differentiation. Cystadenocarcinoma is rare.

Spread

- Direct. The common bile duct is infiltrated early in the course of pancreatic head cancer producing obstructive jaundice. The duodenum, portal vein, and inferior vena cava may be also invaded.
- Lymphatic spread is to adjacent lymph nodes and to nodes in the porta hepatis.
- Blood stream spread to the liver and then to the lungs.
- Transperitoneal spread with peritoneal seeding and ascites.

Clinical features

(A) **Carcinoma of the head**: This tumour manifests relatively early by obstructive jaundice and pruritus. The classic textbook description is that of painless and progressive jaundice in an elderly male. This is, however, not always true as it is sometimes painful, the jaundice may temporarily lessen if an obstructing periampullary carcinoma sloughs, and after all, females are also affected. The jaundice is usually deep olive green. The liver is commonly enlarged because it is engorged with bile, and the gall bladder is palpably distended in half the cases. Anorexia and loss of weight are marked.

(B) **Carcinoma of the body and tail** is late to show its presence. The manifestations are nonspecific, and include epigastric pain radiating to the back, anorexia, loss of weight, and weakness. Migrating inflammation of the superficial veins (thrombophlebitis migrans) is known to accompany visceral malignancies. As the condition is usually late, the liver may be enlarged from metastases, and ascites may be present. Splenomegaly is present in 10% of cases due to splenic vein thrombosis.

Differential diagnosis

Cases with obstructive jaundice should be differentiated from:

1. Calcular obstructive jaundice (Table 33.1).
2. Chronic pancreatitis.
3. Other types of jaundice.

Table 33.1. Differences between calcular and malignant obstructive jaundice

	Calcular	Malignant
Peak age incidence	Middle age	Elderly
Gender	More in females	More in males
Duration of jaundice	May be long	Short
Course of jaundice	Intermittent	Progressive
Depth of jaundice	Moderate	May be very deep
Abdominal pain	Colicky, more in Rt. hypochondrium	May be absent. If present it is dull- aching and referred to back.
Anorexia & loss of weight	Absent	Marked
Fever	May be present	Usually absent
Gall bladder palpation (Fig. 33.12)	Usually impalpable (Courvoisier's law)	Commonly palpable
Ultrasound	Usually fibrosed gall bladder with stones	Marked distended thin-walled gall bladder
CT scan	Gall stones	Head neoplasm is usually seen
ERCP	Stone in CBD	Irregular filling or failure of cannulation of CBD

Investigations

- **Liver function tests** reveal an obstructive pattern of hyperbilirubinaemia (predominant elevation of the conjugated bilirubin), high alkaline phosphatase level, and commonly low prothrombin concentration as a result of nonabsorption of vitamin K which is caused by the lack of bile salts in the intestine.
- **Tumour markers** CA19-9 is useful in patients for whom pancreatic cancer is suspected as a method of confirmation and in follow-up for response to treatment. Carcinoembryonic antigen (CEA) is a tumour marker for gastrointestinal carcinomas is elevated in only 40% to 50% of patients with pancreatic cancer.
- **Abdominal ultrasound** is the first imaging examination to be ordered when obstructive jaundice is suspected. It may fail to show the pancreatic carcinoma because of the overlying colonic gases, nevertheless, the examination is useful to:
  - Document the case as extrahepatic obstructive jaundice by showing dilated extra, and intrahepatic bile ducts.
  - Finds out gall stones which may be the cause of obstruction.
  - Liver metastases are readily diagnosed by ultrasound.
- **CT scan** ideally it should be triphasic, spiral CT with thin cuts (3 mm)
  - Shows the tumour, its local extent, and liver metastases. The tumour classically appears as a hypodense lesion (Fig. 33.13). Of importance is to look for invasion of the superior mesenteric vessels and the portal vein.

Fig. 33.13. CT scan shows carcinoma of pancreatic head and a distended gall bladder.

- It allows targeting the percutaneous fine needle aspiration of the lesion. Biopsy is not needed if clinical evidence, elevated CA 19-9 and a hypodense lesion on triphasic CT are all present. When CT appearance is atypical, needle biopsy is considered. A positive cytology justifies resection of operable cases. On the other hand a negative result cannot rule out malignancy (Fig. 33.14).

- **Endoscopic retrograde cholangiopancreatography (ERCP),** or percutaneous transhepatic cholangiography (PTC) (Fig. 33.15).
  - Exact delineation of the site of obstruction.
  - Differentiate between malignant and stone obstruction of the bile duct.
  - Placing a stent in the bile duct allows drainage of bile to relieve jaundice either as a preparation for surgery, or permanently in very ill inoperable cases.
- **Endoscopic ultrasound.**

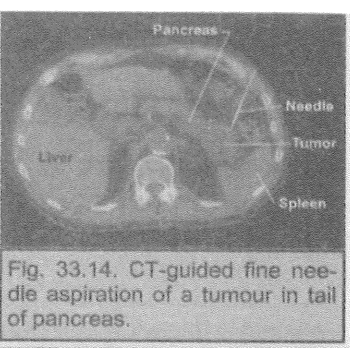
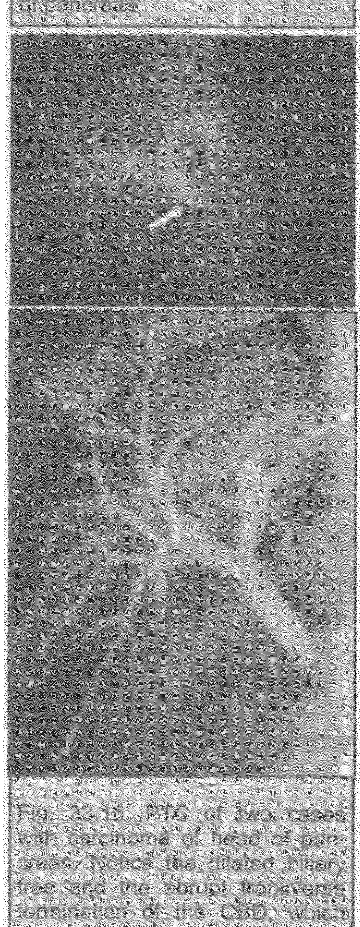


Fig. 33.14. CT-guided fine needle aspiration of a tumour in tail of pancreas.


Fig. 33.15. PTC of two cases with carcinoma of head of pancreas. Notice the dilated biliary tree and the abrupt transverse termination of the CBD, which signifies malignancy.

Treatment

Patients who are unfit for surgery

The very ill jaundiced patients who cannot stand the operation can be treated by an endoscopic stent passed from the duodenum into the common bile duct, and then past the block to the dilated area above it. The stent is left in place to drain the bile into the duodenum to relieve the patient's jaundice. Stenting is, therefore considered a palliative rather than a curative treatment.

Patients who are fit for surgery

Surgery is the best mode of treatment.

Possible complications of surgery on obstructive jaundice:

1. Excessive haemorrhage resulting from hypoprothrombinaemia. Dietary vitamin K is not absorbed because of the lack of bile salts in the intestine, the liver is, therefore, unable to synthesize prothrombin.
2. Septicaemia caused by bacteria migrating from the bowel to the blood stream.
3. Renal and hepatic failure, usually consequent upon the septicaemia.

Preoperative preparation of jaundiced patients aims at avoidance of the mentioned complications.

1. Parenteral vitamin K administration (oral vitamin K is not absorbed), in the form of 10 mg konakion IV bid.
2. The administration of oral bile salts, taken for two days before surgery, is said to prevent migration of bacteria to the blood stream.
3. Prophylactic parenteral antibiotics against gram negative bacilli are given before, and continued during and after the operation.

4. The patient is kept properly hydrated. If the bilirubin is above 15 mg/dl the patient may be given mannitol before, during, and after surgery. This is an osmotic diuretic that is administered intravenously and helps to reduce the incidence of renal failure.
5. Sugar-rich food helps the liver to build up glycogen to stand the stress.

Operation

Exploration is the first step, and upon which depends the choice of procedure.

Operable (potentially curable) cases are those who have neither liver nor peritoneal deposits, and are not fixed to the portal vein. These cases deserve a radical operation, which is pancreaticoduodenectomy, otherwise known as Whipple operation (Fig. 33.16).

The following structures are removed:

- The head and neck of the pancreas.
- The whole duodenum, as it shares the same blood supply as the head of pancreas.
- The antrum of the stomach.
- The gall bladder and the common bile duct.

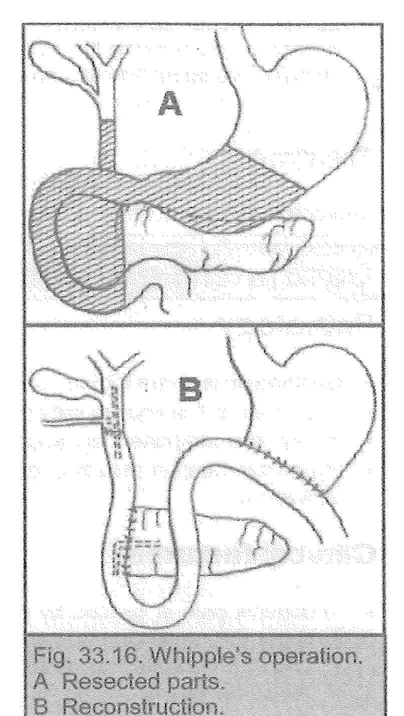


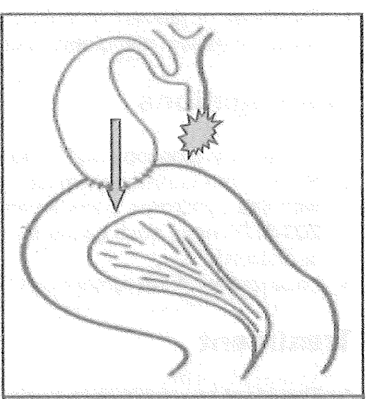
Fig. 33.16. Whipple's operation.
A Resected parts.
B Reconstruction.

The operation ends by performing the following anastomoses:

- The hepatic duct to the jejunum.
- The pancreatic duct to the jejunum.
- The stomach to the jejunum.

Those who are suitable for pancreaticoduodenectomy represent 10-15% of the cases. The operation is a major one with an operative mortality approaching 10%. In addition, the 5-year survival after resection is poor, but it is slightly better with periampullary carcinoma (30%) than carcinoma of the head proper (0-5%).

The majority of cases are inoperable (incurable) either because of liver or peritoneal metastases, or because of invasion of the superior mesenteric and portal veins. These do not deserve a major surgical procedure. Drainage of bile to the intestine is all that can be done. The usual operation is cholecystojejunostomy, i.e., anastomosis of the distended gall bladder to a loop of jejunum (Fig. 33.17). To avoid the risk of future duodenal obstruction by the growing tumour, some surgeons routinely add a gastrojejunostomy.


Fig. 33.17. Cholecysto-jejunostomy is a palliative procedure to relieve jaundice.

Adjuvant chemotherapy, and radiotherapy: may be given after resectional surgery or before operation (neoadjuvant therapy):

- .5-fluoro-uracil and gemcitabine are recommended.

Peptic ulcers are suspected to be due to Z-E syndrome with any of the following conditions:

1. Resistant to treatment.
2. Recurrent.
3. Multiple.
4. In unusual sites.
5. At extremes of age.

Insulinoma

Pathology

- Insulinorna is a tumour of the beta cells of the islets of Langerhans.

- The tumour is usually benign, a few cases are malignant.
- It is commonly found in the body and tail, and is rarely multicentric.
- Some insulinomas are part of multiple endocrine neoplasia (MEN) type I with associated anterior pituitary adenomas and parathyroid hyperplasia.
- The specific feature of insulinomas is the secretion of excess insulin irrespective of the blood glucose level, leading to hypoglycaemia.

Clinical Features

The disease commonly occurs in adults under 40 years of age, who are usually overweight due to over eating. The features are attributable to hypoglycaemia, and are marked when the patient is fasting.

1. Bizarre behaviour and unconscious episodes. Some patients are misdiagnosed as having a psychiatric illness.
2. Palpitation, nervousness, and other symptoms of sympathetic discharge.
3. Whipple's triad
    a. Episodes of the above symptoms are precipitated by fasting or exercise.
    b. During the height of the attack, there is hypoglycaemia below 45 mg/dL.
    c. The symptoms are relieved by glucose.

Investigations

1. The diagnosis is confirmed by demonstrating hypoglycaemia, accompanied by a high insulin level in the blood.
2. Localization is difficult and may be accomplished by CT scan, selective angiography, and by selective venous sampling. The latter Is done by cannulating the splenic vein through the Skin, liver, and then the portal vein. Multiple blood samples are taken from the splenic vein opposite different parts of the pancreas. The sample showing a sharp elevation of insulin level is the one taken from just opposite the tumour.

When operating on obstructive jaundice patients the following complications can possibly take place.
1. Excessive bleeding.
2. Septicaemia.
3. Postoperative renal failure.
4. Postoperative liver failure.

Treatment

Resection of the part of the pancreas containing the tumour. Intraoperative ultrasound is a very useful localization method.

Gastrinoma Zollinger-Ellison syndrome

Pathology

- Gastrinoma is a rare tumour, arising from non-beta islet cells, that secretes excess gastrin hormone.
- In contrast to insulinoma, sixty percent of gastrinomas are malignant.
- It may be a component of multiple endocrine neoplasia (MEN) type I.
- Hypergastrinaemia results in excessive hydrochloric acid secretion, and consequent intractable peptic ulceration.
- Most gasrinoma are found in the gastrinoma triangle (Fig. 33.18)

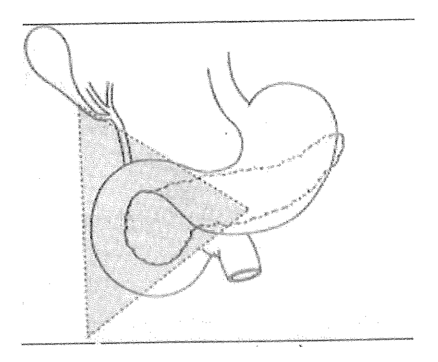


Fig. (33.18): Gastrinoma triangle

Clinical features

- Abdominal pain is caused by peptic ulceration. The ulcers may bleed or perforate. The diagnosis of Zollinger-Ellison syndrome is suspected in the following situations:

- Ulcers are resistant to treatment with H2 receptor antagonists.
- Recurrent ulcers.
- Multiple ulcers, and ulcers in unusual sites as the distal duodenum and proximal jejunum.
- Peptic ulcers at the extremes of age.

- Diarrhoea is present in a third of cases because the hyperacidity inactivates pancreatic enzymes, and because gastrin stimulates intestinal motility.

Investigations

1. Upper GI endoscopy and barium meal examination show the ulcers.
2. Radioimmunoassay estimation of serum gastrin. The normal level is usually below 150 ng/L. Most cases with this syndrome show levels more than 500 ng/L. Some patients have intermediate levels between 200-5000 ng/L, such cases show doubling of the serum levels when stimulated with intravenous calcium or secretin.
3. Localization of the tumour by CT scan, selective arteriography, and selective venous sampling.
4. Endoscopic ultrasound and ^{111}In octreotide scintigraphy.

Treatment

1. The ideal treatment is to excise the part of the pancreas containing the tumour. Only 20% of tumours are suitable for resection because of multiplicity, and poor localization. Intraoperative ultrasound is very useful in localization.
2. Usually tumour resection is not possible, and total gastrectomy is done to completely abolish acid secretion.
3. A less effective alternative is prolonged intensive therapy with proton pump inhibitors.

SPLEEN

Anatomy

The normal spleen varies in weight from 80-300 g. It is an intraperitoneal structure with a notched anterior border. The convex parietal surface of the spleen is in contact with the diaphragm deep to the 9th, 10th and 11th ribs. Its long axis follows the 10th rib down to the rnidaxillary line. Through the diaphragm the spleen is related to the pleural recess and to the thin inferior border of left lung (Fig. 34.1).

The visceral surface of the spleen is related to the stomach, left kidney and colon. The hilum of the spleen is related to the tail of pancreas.

CHAPTER CONTENTS

Splenic ligaments

The spleen is completely covered with peritoneum which is fixed firmly to the splenic capsule. The spleen is suspended at its hilum by two peritoneal folds, the lieno-renal and the gastrosplenic ligaments (Fig. 34.2).

1. **The lieno-renal ligament** is short and transmits the blood vessels to the spleen, the tail of the pancreas lies in this ligament.
2. The gastrosplenic ligament contains the short gastric arteries which are branches of the splenic or left gastroepiploic artery and which supply the left half of the greater curvature and the fundus of the stomach.
3. The phrenicocolic ligament attaches the splenic flexure of the colon to the diaphragm. The lower pole of the spleen is in contact with this ligament which is thought to be one of its main supports.

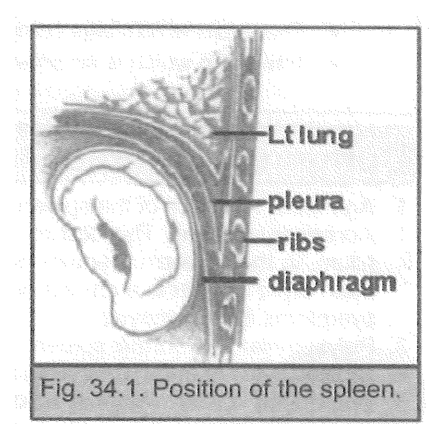


Fig. 34.1. Position of the spleen.

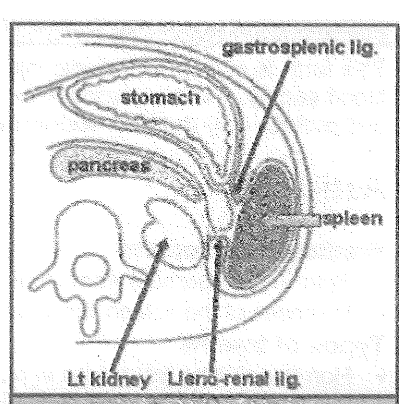


Fig. 34.2. Transverse section in the abdomen showing the peito-neal folds that are related to the spleen.

Blood supply

Splenic artery

This is usually a branch from the celiac artery but it may arise from the aorta or the superior mesenteric artery. It passes along the upper border of the pancreas.

At the tail of the pancreas it passes to the front of it, and divides into its superior and inferior terminal branches that enter the splenic hilum.

Splenic vein

It runs along the posterior surface of the pancreas below the level of the splenic artery.

It receives the superior mesenteric vein at a right angle behind the neck of the pancreas to form the portal vein.

Lymphatic drainage

The capsule and large connective tissue trabeculae are provided with lymphatic vessels that drain into lymph nodes at the hilum.

Physiology

Functions of the spleen

1. **Fetal erythropoiesis**. During the 5th to 8th month of foetal life the spleen shares in the production of both red and white cells. This function does not continue in the normal adult. If the bone marrow production of red cells becomes defective after birth, splenic erythropoiesis will continue, e.g., in myeloflbrosis.
2. **Sequestration of old blood cells**. Aged and abnormal erythrocytes, abnormal granulocytes, normal and abnormal platelets and cellular debris will be cleared by the spleen, which is capable of discrimination between these and normal cellular components. The platelets (thrombocytes), under normal conditions, survive about 10 days in the circulation. One third of the normal platelet pool is normally sequestered in the spleen. In splenomegaly up to 80% of platelets are sequestered in the spleen and this may lead to thrombocytopenia,
3. **Defensive functions:**
    a. Reticuloendothelial macrophages of the spleen are capable of destroying foreign substances by the process of phagocytosis. These foreign substances include bacteria, fungi, protozoa and particulate matter. Also phagocytosis of abnormal lipoid is the cause of splenomegaly in lipoid dystrophies,
    b. he spleen plays an important role in proliferation of T-lymphocytes and the antibody forming B lymphocytes.
    c. It helps in the production of various antibodies including:
        i. IgM.
        ii. Tuftsin which stimulates phagocytosis by neutrophils.
        iii. Opsonins which react with bacteria and fungi to make them more susceptible to phagocytosis.
        iv. Properdin which fixes complement to bacterial and fungal surface prior to phagocytosis.
        v. Interferon which is an antiviral antibody.

Congenital anomalies

1. Agenesis: Absence of the spleen (asplenia) is very rare.
2. Accessory spleens. This occurs in 10-35% of individuals. The majority of accessory spleens occur at the hilum, in the greater omentum or along the splenic vessels and pancreas. If the spleen is removed for haemolytic anaemia, a careful search must be made for accessory spleens which can cause recurrent symptoms if not removed.
3. Polysplenia: The normal spleen is deeply lobulated or divided into two or more parts.
4. Splenic cysts of embryonal rests include dermoid and mesenchymal inclusion cysts.
5. Wandering spleen (Splenic ectopia). This anomaly is caused by laxity of gastrosplenic and splenophrenic ligaments, or defects in their development which permits movement of the spleen in the abdomen. Torsion of the pedicle is the most frequent complication.

Rupture of the spleen

This term is applied to splenic injuries in which there is disruption of the orga parenchyma, capsule or blood supply. It is a common accident in middle aged males. T spleen is normally small, hidden by ribs and protected by the thick abdominal muscles.

Aetiology

Predisposing factors

- Splenic enlargement which makes it more liable to trauma.
- Diseases of the spleen like malaria which make it soft and more friable.

Types of trauma

- Non penetrating trauma as in blunt abdominal trauma or trauma to lower thora cage. This is usually a result of road traffic accidents and falling from a height.
- Penetrating trauma as a consequence of gunshots or stabbing.
- Operative trauma occurs during an operation on adjacent viscera, e.g. during gas or colonic surgery. Injuries usually result from retractors placed against the organ order to obtain good exposure.

Spontaneous rupture of the spleen is rare.

Pathology

Types of splenic injury

1. Subcapsular haematoma.
2. Small superficial tears single or multiple.
3. Deep tear, single or multiple and reaching the hilum.
4. Avulsion of a pole of the spleen.
5. Complete pulping of the spleen.
6. Injury of the vascular pedicle, i.e. avulsion or thrombosis of the vessel.

Complications

1. Haemorrhage which is usually internal. Blood may accumulate in the periton cavity, retroperitoneal space, or in the pleura if the injury involves the diaphrag
2. Associated other abdominal or thoracic injuries.
3. Splenic cyst may follow a perisplenic haematoma.

Clinical picture

There are 3 clinical types of rupture of the spleen;

1. The fatal type.
2. Classical rupture.
3. Delayed rupture.

Fatal type

The tear is deep or the pedicle is ruptured and haemorrhage is so massive that t patient is severely shocked with rapid death occurring before any surgical intervention.

Classical rupture

This is the commonest presentation

- The general manifestations of internal haemorrhage are present with increasing pall weak and rapid pulse, low blood pressure and air hunger due to oxygen lack.

- Abdominal examination shows tenderness and rigidity in the left hypochondrium which spreads into the abdomen and evidence of shifting dullness. Distension is present due to intestinal paresis.
- Special signs may be present in rupture of the spleen but they are not essential for the diagnosis.
    - **Balance's sign**. Shifting dullness on the right side and fixed dullness on the left side. This is due to free fluid blood in the peritoneal cavity in the right flank, but on the left side the fixed dullness is due to blood clots in the peritoneal cavity, and also due to retroperitoneal haematoma.
    - **Kehr's sign**. The patient has pain in the left shoulder, this is referred pain due to irritation of the diaphragm. This pain occurs especially if the patient is put in the Trendlenburg position. Also pressure on the left hypochondrium leads to referred pain in left shoulder.
    - **Cullen's sign**. Brownish or bluish discoloralion around the umbilicus may occur in about 20% of people who have thin linea alba around the umbilicus through which blood can shine.

Delayed rupture

The initial shock is followed by a long lucid interval, which may be few days or weeks. About two weeks after the accident, which may be forgotten, the patient presents with the picture of internal haemorrhage.

This delay of clinical presentation may be due to:

1. The formation of subcapsular haematoma which may rupture later.
2. The greater omentum seals the region of the spleen from the general peritoneal cavity, and then retracts releasing blood.
3. A clot may form to block the tear and stop bleeding and is later on dislodged when the blood pressure rises or is digested by enzymes from the injured pancreas.

Management

Follow the guidelines of trauma life support that are described in chapter 2.

Investigations

1. **Blood picture**. Declining haemoglobin and haematocrit values denote haemorrhage.
2. **Ultrasound or CT scan** (Fig. 2.11) of the abdomen. The spleen is visualized with a surrounding haematoma. Serial examinations will diagnose an enlarging haematoma. Free blood in the peritoneal cavity is detected. Ultrasound and CT scanning have diagnostic accuracy more than 90%.
3. **Plain x-ray** of the upper abdomen raises suspicion of rupture of the spleen by the following radiological signs:
    a. Obliteration of psoas shadow.
    b. Fracture of one or more of lower ribs.
    c. Elevation of left side of diaphragm.
4. **Peritoneal lavage** reveals blood.

N.B. If the patient is in severe shock, there is no need for investigations. The surgeon depends on clinical findings and proceeds for immediate laparotomy to stop bleeding.

Treatment

One of 3 options will be followed:

(A) A minor splenic injury in a haemodynamically stable patient can be treated conservatively by rest in bed, resuscitation and meticulous follow-up for up to 7-10 days.

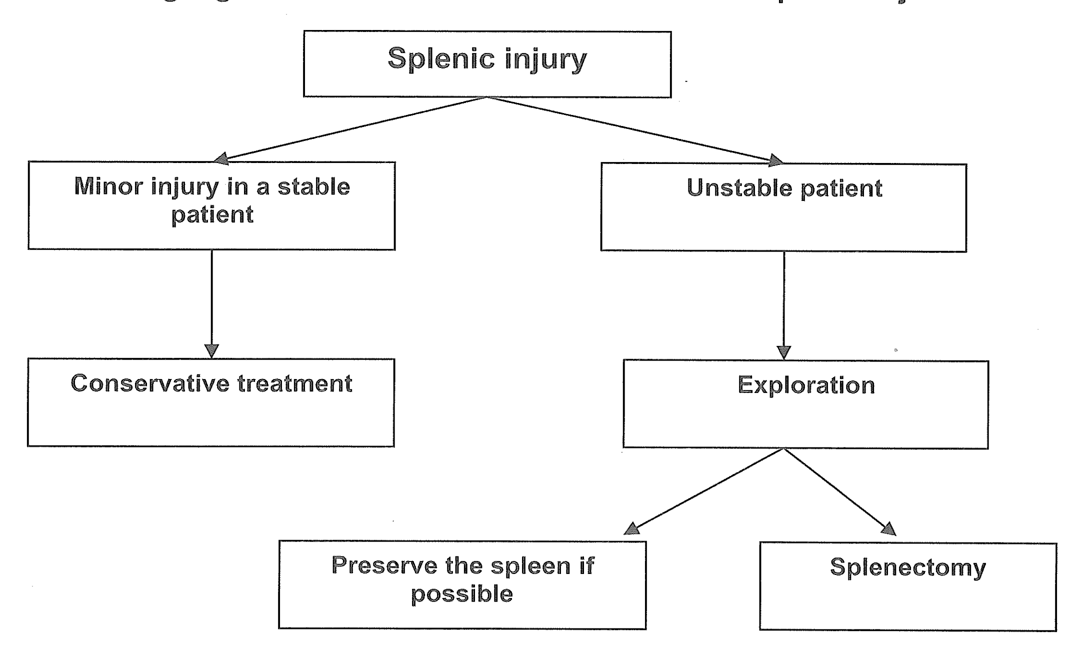
All unstable patients need urgent laparotomy after correction of hypovolaemia.

(B) Splenectomy should be performed if there are deep lacerations or avulsion of the splenic pedicle. If splenectomy is performed in a young child, it is necessary to administer anti-pneumococcal vaccine (pneumovax) every 5 years until the age of 18 years.

(C) In children especially below the age of 15 years every attempt should be made to preserve the spleen due to the vital role it plays in the immune mechanism in this age group. Several techniques are available including:

a. Suture of a small laceration.
b. Partial splenectomy.
c. Compression of a lacerated spleen in a polygalactin (vicryl) mesh. After conservative surgery meticulous observation is necessary in the post-operative period as the patient may rebleed.

The following algorithm summarizes the treatment of splenic injuries



Infarction of the spleen

Aetiology

- May be spontaneous in patients with splenomegaly,
- Vascular occlusion produced by sickle cell disease.
- Embolism from infected heart valve or bacterial endocarditis.

Clinical features

- Splenic infarction may be asymptomatic.
- It is commonly accompanied with left upper quadrant abdominal pain.
- Overlying guarding. A friction rub may be heared over splenic area.

Treatment

1. The treatment is by rest and analgesics.
2. Sometimes septic infarcts cause an abscess which needs splenectomy.

Infections of the spleen

1. **Acute abscess**: This is a rare condition occurring as a sequel of specific fever, e.g. typhoid fever, It can also be blood borne from a distant septic lesion or during systemic pyaemia. Percutaneous drainage under ultrasound or CT guidance is a suitable line of treatment as splenectomy may be very difficult due to dense adhesions.
2. **Chronic abscess**: This is also rare and can result form a neglected acute abscess or an infected cyst. Treatment is splenectomy.
3. **Tuberculosis**: The disease is usually a manifestation of generalized miliary TB and rarely a primary affection. Multiple areas of caseation occur.
4. **Protozoal** infection of the spleen occurs in malaria and kala azar.

Bilharziasis of the spleen

Aetiology

The condition is caused by infection with *Schistosoma mansoni* in 75% of cases and by Schistosoma haematobium in 25% of cases.

Causes of splenic enlargement in Bilharziasis

- In the early stages the splenic enlargement results from reticulo-endothelial hyperplasia due to:
  - Absorption of toxins from the living worms and ova.
  - Deposition of ova in the splenic radicles
- With the advent of hepatic fibrosis, chronic venous congestion follows and leads to progressive enlargement of the spleen (congestive splenomegaly).

Pathology

Gross picture

- The spleen is enlarged with a thick capsule.
- Subcapsular haemorrhages and patches of perisplenitis, due to organized haematomas or infarcts occur.
- Thick vascular adhesions may bind the capsule to the diaphragm and nearby structures.
- The splenic vessels are markedly enlarged and tortuous.
- On section the cute surface is firm in consistency and red in colour with prominent lymphoid follicles and thick greyish trabeculae.

Histologically

- The red pulp is congested and infiltrated with eosinophils.
- The lymphoid follicles are enlarged and may show germinal centers.
- The littoral cells of the sinusoids and the reticular cells of the pulp proliferate and show active phagocytosis.

Complications

Susceptibility to injury.
Susceptibility to infarction.
Hypersplenism.

Clinical features

It is to be noticed that most of the complaints in patients with Egypt hepatosplenomegaly are secondary to two pathological problems, which are liver (insufficiency and portal hypertension (Chapter 31).

The spleen itself is not responsible for most of the troubles of the patient, ap from:

1. It causes some dragging pain if it is very large.
2. It splenic infarction occurs; it produces pain due to peritoneal or diaphragma irritation.
3. Splenic enlargement may lead to secondary hypersplenism. To prove the preser of hypersplenism the peripheral blood picture should reveal anaemia, leucoper thrombocytopenia or (pancytopenia) and bone marrow aspiration should reveal t presence of hypercellularity. More confirmation is obtained by injecting the patier own RBCs after being tagged with radioactive ^{51}Cr and estimating the half life of t RBCs. Patients with hypersplenism will show diminished half life of RBCs and t spleen/liver radioactivity index is increased.

Treatment

Splenectomy in patients with Egyptian hepatosplenomegaly is indicated only in t following conditions:

1. Hypersplenism.
2. Huge splenomegaly which is cumbersome to the patient.
3. Recurrent attacks of pain secondary to splenic infarctions.
4. As a part of operations done for portal hypertension as portoazygos disconnection splenorenal shunt.

Hydatid Disease may lead to the formation of single or multiple cysts of the splee Treatment is splenectomy.

Malarial Spleen is common in endemic areas of malaria. The spleen may reach huge size and is soft. It acts as a reservoir for malaria parasites and every now and then causes an attack of malaria. Malarial spleen is very liable to rupture as it is sc Splenectomy is more difficult because the organ is friable.

Kala Azar is characterized by hepatosplenomegaly with progressive emaciatic Blood examination reveals Leishmania donovan.

Cysts of the spleen

Splenic cysts are rare.

- May be multiple where cystic disease of the spleen is usually associated with cys disease of liver, kidney lung and pancreas.
- May be single as hydatid cyst, lymphatic cyst, blood cyst or cystic degeneration tumours. Treatment is by splenectomy.

Tumours of the spleen

Splenic tumours are not common. They may be:

1. Part of generalized reticuloendothelial affection as in malignant lymphomas a leukaemias.
2. A tumour that is confined to the spleen
   a. Benign tumours as lymphangioma and cavernous haemangioma.
   b. Primary malignant tumours as localized forms of lymphoma a fibrosarcoma.

c. Secondary tumours. These are very rare as it is supposed that the reticulo-endothelial cells can destroy the secondary malignant cells when they reach the spleen. The spleen may be infiltrated by direct extension from carcinoma of the stomach or pancreas.

Splenomegaly

The normal spleen cannot be palpated clinically. If the spleen is palpable under the costal margin, it is at least 3 times the normal size. Enlargement of the spleen varies from being just palpable to a huge size which may reach below the level of the umbilicus down to the right iliac fossa.

Causes of splenomegaly

1. Infections
   a. Bacterial: Typhoid and paratyphoid, tuberculosis, typhus, anthrax, abscess and syphilis.
   b. Viral: Infective mononucleosis and psittacosis.
   c. Parasitic: Schistosomiasis and hydatid cyst.
   d. Protozoal: Kala azar and malaria.
2. Blood diseases
   a. Haemolytic anaemias,
   b. Leukaemias.
   c. Thrombocytopenias.
   d. Myelofibrosis.
   e. Polycythaemia vera.
3. Metabolic causes, Gaucher's disease, porphyria and rickets.
4. Collagen diseases. Felty's syndrome and Still's disease.
5. Portal hypertension.
6. Cysts of the spleen.
7. Tumours of the spleen.

Clinical characters of splenic enlargement

1. An intra-abdominal swelling in the left hypochondrium that may descend downwards and medially towards the umbilical region.
2. It moves up and down with respiration.
3. It has a sharp medial border with a notch.
4. The consistency varies from soft (typhoid and malaria) to firm (bilharziasis) to hard (lymphomas and leukaemia).
5. The surface is usually smooth. A nodular spleen may be seen in some cases of lymphomas.
6. The fingers cannot be insinuated between the spleen and costal margin so the upper border cannot be reached.
7. The splenic swelling does not fill the loin and does not ballot.
8. Percussion reveals dullness anteriorly and resonance posteriorly (the lung passes down behind the spleen).

Differential diagnosis of a huge splenic enlargement

- Thalassaemia in children.
- Myelofibrosis.
- Portal hypertension.
- Gaucher's disease in children.
- Malaria.

Haemolytic anaemias

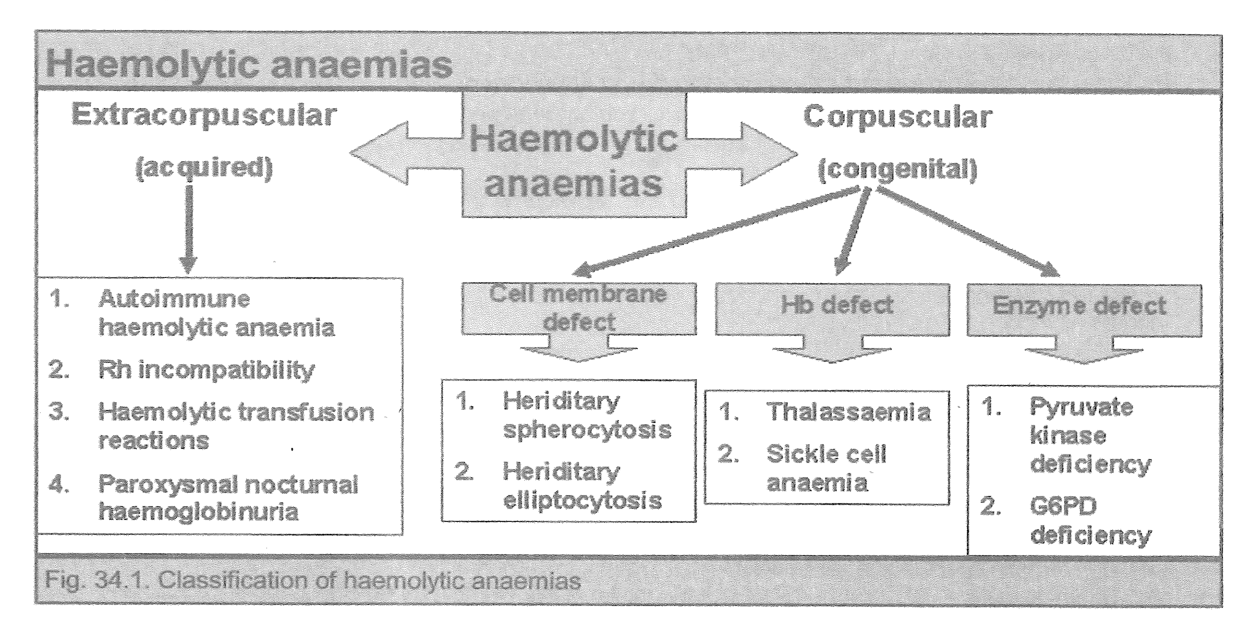


Fig. 34.1. Classification of haemolytic anaemias

This category includes a wide spectrum of disorders in which there is accelerated destruction of mature red blood cells.

Haemolytic anaemias are generally classified as congenital or acquired (Fig. 34.1).

- Congenital anaemias are due to an intrinsic abnormality of the red blood cells.
- Acquired anaemias are related to extracorpuscular factors acting on normal red blood cells.

In haemolytic disorders the reduced red blood cell survival may be demonstrated by measuring the disappearance of the patient's radioactive red blood cells (labelled with ^{51}Cr) and the splenic role may be evaluated by determining the relative uptake of this radioactivity by the spleen and liver.

Of interest to the surgeons are the following types of haemolytic anaemias:

1. Haemolytic anaemias that are treated by splenectomy.
    a. Heriditary spherocytosis is cured by splenectomy.
    b. Some cases of thalassaemia require splenectomy, which is less beneficial than in spherocytosis.
    c. Rarely sickle cell anaemia or autoimmune types.
2. Sickle cell anaemia patients develop acute attacks if exposed to hypoxia. If they require surgery for any reason, the anaesthetist should ensure adequate oxygenation all through the operation.

Heriditary spherocytosis will be discussed as an example of haemolytic anaemias.

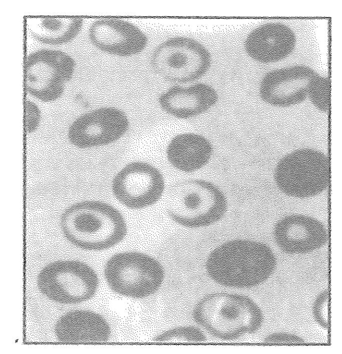
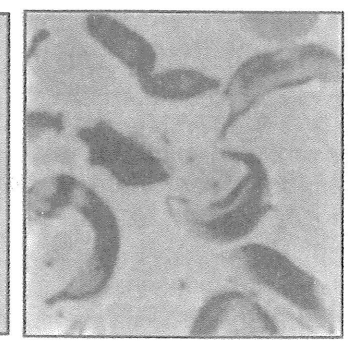


Fig. 34.2. Appearance of RBCs on a blood film:

Left: Target cells of Thalassaemia.

Right: Sickle cells of sickle cell anaemia.



Immune thrombocytopenic purpura (ITP)

Aetiology

This disease was formerly known as idiopathic thrombocytopenic purpura. It is now known to be an autimmune disease. Platelets are sensitized with an auto-antibody that results is their early sequestration by the reticuloendothelial cells (mainly the spleen and liver).

Pathology

The term ITP should be reserved for the haen,orrhagic disorder characterized by:

1. Subnormal platelet count.
2. Bone marrow contains normal or increased megakaryocytes.
3. No systemic disease or injection of drugs capable of inducing thrombocytopenia.
4. The spleen is implicated either as a source of antibody production or as a major destruction site for sensitized platelets.

Clinical features

Symptoms

- The disease occurs most commonly in children and young adult females.
- The course of the disease runs in remissions and relapses.
- It may present by an attack of bleeding under the skin (ecchymosis) or mucous membrane or from an orifice, as the nose, urinary or gastrointestinal tract. Menorrhagia is a common complaint in affected young females. Intracranial hemorrhage is rare, but is the most frequent cause of death.

Physical examination shows

- Pallor.
- Cutaneous ecchymoses.
- Slight enlargement of the spleen. A large spleen is against the diagnosis of ITP.
- Capillary resistance test of Hess is positive.

Investigations

- The red and white cells are diminished due to repeated haemorrhages.
- Platelets are diminished in number. Their count may drop to 30.000/ul.
- Bleeding time is prolonged up to 20 minutes (normally 2-5 mm). Coagulation time is normal.
- The blood clot is soft, friable and fails to retract.
- Bone marrow examination reveals a large number of megakaryocytes, but there is failure of budding.

Treatment

- In children short courses of corticosteroids or azathioprine are usually followed by recovery. In adults the disease relapses and becomes more severe.
- Splenectomy is indicated if ITP manifestations persist for more than 9 months. A response to steroids usually predicts a good response to splenectomy.

Differential diagnosis of thrombocytopenia:

1. Diminished production by the bone marrow in patients with aplastic anaemia or secondary to cytotoxic drugs.
2. Increased platelet consumption in disseminated intravascular coagulation.
3. Increased platelet destruction by the spleen.
   - Immune thrombocytopenic purpura.

Hereditary spherocytosis

Pathology

Inheritance

The disease is transmitted as Mendelian autosomal dominant. Males and females are equally affected.

Pathogenesis

- There is a defect in the red-cell membrane due to deficiency of certain proteins known as spectrins.
- As a result, there is excessive permeability to sodium ions and the red cell integrity can only be maintained by increasing the glycolytic activity to provide the energy required to pump out the sodium ions.
- The increased metabolic activity leads to the development of microspherocytosis in the red pulp where the available glucose is diminished.
- Microspherocytes are trapped in the red pulp because they lack deformability and so they are subsequently destroyed.

Complications

- Choledocholithiasis. Pigmented gallstones may occur in up to 60% of patients. They may occur under the age of 10.
- Rarely chronic leg ulcers may develop. They may be bilateral.

Clinical features

- The disease may present at early infancy or it may be delayed.
- There is mild haernolytic jaundice accompanied by mild to moderate anaemia.
- The spleen is enlarged.
- Sometimes, hepatomegaly is also present.
- The patient is liable to recurrent attacks of haemolytic crisis, which may be precipitated by infection. During the attack there is severe pallor, pyrexia, abdominal pain, and vomiting, followed by jaundice.

Investigations

1. There is anaemia, the red cell count is diminished. RBCs are small and biconvex (microspherocytes).
2. The reticulocytic count is increased up to 5-20%.
3. Fragility test. This test is characteristic. Normally haemolysis starts at 0.45% saline solution. In hereditary spherocytosis, haemolysis starts at 0.6%.
4. Indirect serum bilirubin is increased.
5. Radioactive studies using ^{51}Cr utilizing the patient's own RBCs will reveal shortened half life and increased radioactivity over the spleen.
6. Abdominal ultrasound, may reveal gall stones.

Treatment

- Splenectomy is curative. Although the RBCs remain abnormal, their life span is increased.
- The best timing of the operation is at the age of 6-7 years. Before this age there is the risk of increased liability to infection due to the loss of the immune mechanism of the spleen. If surgery is postponed after this age, the chance of developing gallstones is increased.
- During surgery meticulous search for accessory spleneculi is important to avoid recurrence of hemolytic attacks. Should the patient have gallstones, cholecystectomy can be performed simultaneously.

- Secondary to drug intake or certain infections.
- Hypersplenism.

Hypersplenism

Hypersplenism means that the spleen is overdoing its function of getting rid of old blood cells. It, therefore, destroys also young healthy blood cells. The result is pancytopenia (anaemia, leucopenia and thrombocytopenia). The bone marrow becomes hyperactive in an attempt to compensate for the low blood cell counts.

Primary hypersplenism

This is the idiopathic type of the disease. The aetiology has not yet been determined but ^{51}Cr tagged red cell sequestration studies indicate that the spleen is the major site for phagocytosis. In some patients lymphoproliferation of the spleen is so marked as to suggest the development of premalignant lymphoma.

Clinical features

The majority of patients are females.

- Fever, frequent infections and oral ulceration may be noticed with neutropenia.
- Petichae and ecchymosis are due to thrombocytopenia.
- Pallor indicates destruction of red cells.

Investigations

- Blood picture reveals low counts of RBC5, leucocytes, and platelets; with elevated reticulocyte count. One type of blood cells may be affected more than the others.
- Peripheral blood smear is devoid of any diagnostic features of leukaemia or myeloproliferative disease.
- Bone marrow reveals pancellular hyperplasia.

Treatment

Splenectomy helps haematologic improvement in ITP patients.

Secondary hypersplenism

Pancytopenia may arise from portal hypertension. Splenomegaly with congestion of the vascular spaces accompanies portal hypertension and accelerates destruction of the circulating cells within the spleen.

Clinical features

- Anaemia, pallor and increased rate of infection are obvious.
- Petichae and spontaneous bleeding are rare.

Treatment

- Splenectomy is required for severe cases. This may be combined with shunt surgery or portoazygos disconnection to decompress the oesophageal varices,
- Percutaneous transfemoral arterial embolization of splenic artery may control splenic hyper-function, but the development of painful infarctions of the spleen and septic Splenitis limit this line of treatment.

Splenectomy

Indications

1. Traumatic injury of the spleen.
2. Haematological diseases

- Hereditary spherocytosis.
- Some patients with acquired haemolytic anaemia.
- Immune thrombocytopenic purpura (ITP).
- Some patients with thalassaemia who require frequent blood transfusions, particularly if they have acquired haemolytic antibodies.
- Few patients with sickle cell anaemia.

3. Lymphomas. Splenectomy may be performed for staging Hodgkin's disease, and to reduce the dose of radio or chemotherapy.
4. Splenectomy is included during radical surgery for the stomach, esophagus or pancreas.
5. Splenectomy and devascularization and lieno-renal shunts are performed for oesophageal varices.
6. Hypersplenism.
7. Splenic cysts.
8. Splenic abscess.
9. Splenic tumours.

Procedure

- General anaesthesia.
- Supine position.
- Left para median or left subcostal incision.
- The left hand is placed behind the spleen which is drawn downwards and medially to divide the posterior leaflet of the lienorenal ligament and any adhesions to the diaphragm.
- The spleen is delivered through the incision, the lower pole first, followed by the upper pole.
- The short gastric vessels are ligated and divided followed by the left gastroepiploic vessels.
- Gentle dissection is done to separate the tail of the pancreas from the splenic vessels.
- The splenic artery and vein are separated, clamped, divided and doubly ligated taking care not to injure:
    - The tail of the pancreas.
    - The splenic flexure of the colon.
    - The left kidney and adrenal.
- Splenectomy is completed by dividing the residual peritoneal attachment to the stomach and colon.
- Good haemostasis after peritoneal wash with warm saline. A drain may be left if there is doubt about absolute haemostasis.
- The incision is closed in layers.

Complications

(A) **Complications that are common to all operations**

a. Reactionary haemorrhage if a ligature slips off an artery or a vein.
b. Atelectasis and pneumonia. Upper abdominal incisions are painful and restrict depth of respiration.
c. Deep vein thrombosis.
d. Surgical site, burst abdomen, and incisional hernia are uncommon.

(B) **Specific complications of splenectomy**

a. Acute gastric dilatation following mobilization of the stomach to ligate the short gastric vessels.

b. Portal vein thrombosis because splenectomy is frequently followed by rise in white blood cells and platelets.
c. Pancreatic fistula may follow damage to the tail of the pancreas during mobilization of the splenic pedicle. This condition may be accompanied by left pleural effusion, peritoneal effusion, or abdominal wound dehiscence.
d. Gastric fistula due to injury of the wall of the stomach during ligation of the short gastric vessels.
e. Haematemesis may occur due to congestion of the gastric mucosa after ligating the short gastric vessels.
f. Subphrenic haematoma or abscess.
g. Post-splenectomy bacterial infections are likely to occur because the spleen phagocytoses bacteria specially the encapsulated type. Patients with splenectomy are susceptible to infections produced by Streptococcus pneumonia, Neisseria meningitides and Haemophilus influenzae. The risk is higher if splenectomy is done in childhood. A serious form is overwhelming post-splenectomy sepsis (OPSI) which is fatal. Therefore, splenectomized children should receive anti-pneumococcal vaccine (pneumovax), meningococcal vaccine and anti haemophilus influenza vaccine until the age of 18 years. Patients should commence Amoxil at first sign of febrile illness.

PERITONEUM, OMENTUMAND MESENTERY

CHAPTER CONTENTS

The peritoneum; anatomical facts

Parts

The peritoneum is a single layer of flat mesothelial cells resting on a bed of loose connective tissue, It is divided into 2 two parts:

The parietal peritoneum is the part that lines the entire abdominal cavity, covering the inner surfaces of the abdominal wall and pelvis. It is reinforced by the fascia transversalis, which lies external to it.

The visceral peritoneum covers all the intra-abdominal viscera and mesenteries.

Peritoneal pain

Parietal peritoneum is highly sensitive to pain stimuli.
Stim → Sharp localized pain.

Visceral peritoneum is relatively insensitive.
Stim → Dull ill-localized pain

Innervation

The parietal peritoneum is sensitive and is innervated by both somatic and visceral afferent nerves. The anterior parietal peritoneum is the most sensitive and any local insult to the parietal peritoneum leads to protective voluntary muscle guarding and then reflex muscular spasm. The visceral peritoneum receives innervation only from the autonomic nervous system and is relatively insensitive.

Healing

When parietal peritoneal defects are created, healing occurs not from the edges but by metamorphosis of in-situ mesenchymal cells, large defects heal as rapidly as small defects.

Generalized septic peritonitis

Inflammation of the peritoneal cavity by pyogenic organisms is a fife- threatening problem.

Aetiology

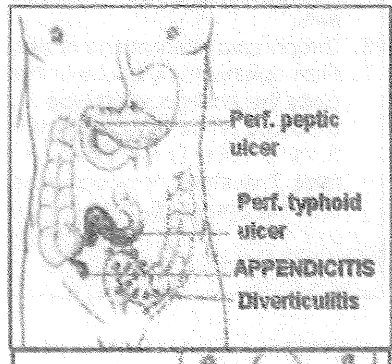
Causative organisms include:

E. coli, aerobic and anaerobic streptococci, bacteroids, and pneumococci.

Sources of infection (Fig. 35.1)

Organisms reach the peritoneal cavity by:

- Local spread from:
  1. Infected organs, e.g. appendicitis, cholecystitis or diverticulitis.
  2. Leaking organs, e.g. perforated peptic ulcer, perforated typhoid ulcer, leaking anastomosis, ruptured gut or extravasated urine.



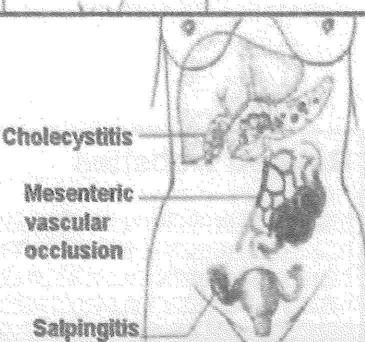


Fig. 35.1. Common causes of peritonitis + leaking anastomosis.

This route represents the commonest source of infection.

- Direct entry through, an operative or traumatic wound.
- Blood spread may occur in cases of septicaemia and pyaemia, but is rare.
- Primary peritonitis: This is an uncommon form of septic peritonitis that occurs with no apparent intraperitoneal pathology, It usually affects female children where infection is thought to reach the peritoneal cavity through the fallopian tubes. Pneumococci and streptococci are the main pathogens.

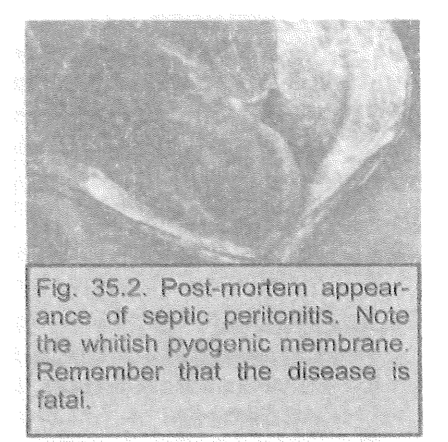
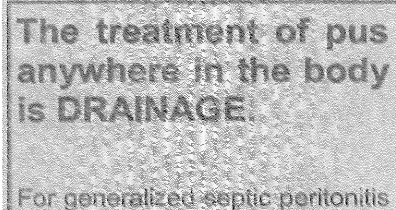


Fig. 35.2. Post-mortem appearance of septic peritonitis. Note the whitish pyogenic membrane. Remember that the disease is fatal.



The treatment of pus anywhere in the body is DRAINAGE.

For generalized septic peritonitis urgent laparotomy is done to achieve:

1. DRAINAGE of pus.
2. Control of the source, e.g., removal of ruptured appendix or plugging of a perforated peptic ulcer.

Pathology

Gross changes

1. Inflamed areas become opaque and adhere together because of fibrin deposition (Fig. 35.2).
2. Excess purulent exudate accumulates.
3. Paralytic ileus occurs at first as a reflex to minimize spread, and is then accentuated by the toxic effect of pus. Finally mechanical intestinal obstruction from fibrinous adhesions may complicate the picture.

Fate

The fate depends on the virulence of the organisms on one side and the efficiency of treatment and the body resistance on the other:

Resolution. The peritoneum has great resistance to infection. If the source of infection is controlled or removed, peritonitis resolves rapidly.

Localization (abscess formation). If complete resolution fails, pus localizes to form an abscess which may be local or remote:

- Localization may occur around the primary focus (e.g., in acute appendicitis or cholecystitis) by adhesions of intestine and omentum around the inflamed organ to form an abscess.
- Localization may occur in one of the anatomical compartments of the peritoneal cavity to form a remote abscess, e.g. a subphrenic, iliac or pelvic abscess depending on the position.

Flaring up. This causes generalized peritonitis. The factors which predispose to it include:

- High virulence of the organisms.
- Sudden perforation of a hollow viscus which does not allow the defensive mechanisms to localize the source of infection.
- Persistent source of infection.
- Stimulation of peristalsis by eating, enemas or purgatives.
- Rough handling of a localized collection during surgery.
- Immunosuppression as in diabetes mellitus, AIDS, and in patients receiving corticosteroids or chemotherapy.
- Generalized peritonitis is likely to occur in children because the greater omentum, which has an important role in localizing inflammatory processes, is small in size and note well developed.

Seticaemia, multiple organ failure (MOF), and death.

Clinical features

The patient presents at first by the clinical picture of the original cause, e.g. ac appendicitis or acute cholecystitis. When the condition is fully established it may be diffi to tell the original cause.

Symptoms

1. Pain is persistent, dull aching, and is increased by movements or coughing. site of maximum pain is usually at the site of the original lesion.
2. Vomiting. At first gastric contents are vomited then the vomitus becomes bilious long standing cases the vomitus becomes faeculent.
3. Abdominal distension.
4. Absolute constipation. If there is a pelvic collection the patient may have tenesr or diarrhoea.

Signs

1. The patient looks distressed and toxic. There is pyrexia and tachycardia. patient lies still in bed avoiding any movements.
2. Abdominal examination reveals generalized tenderness, rebound tenderness rigidity. The site of maximum tenderness and rigidity indicates the aetiology, e.g the signs are maximum at the right iliac fossa, it denotes that peritonitis secondary to perforated appendicitis.
3. Auscultation reveals absent bowel sounds due to paralytic ileus.
4. In neglected cases the patient will present by sunken eyes and an anxious l (Facies hippocratica). The clinical picture of septicaemic shock supervenes.

Investigations

1. Blood picture. Polymorphnuclear leucocytosis with shift to left are always present
2. Plain abdominal X-ray:
   - It demonstrates paralytic ileus with distension of the small and large bowel.
   - It may determine the primary lesion, e.g. perforated peptic ulcer shows air un the diaphragm.
3. Abdominal ultrasound will reveal free fluid in the peritoneal cavity.
4. Peritoneal diagnostic aspiration may be helpful in determining the nature of fluid.

Treatment

Acute generalized peritonitis is an emergency because it denotes failure of defensive mechanisms to localize the infection.

Preoperative preparation

1. A naso-gastric tube is inserted to deflate the stomach and bowels and to prev vomiting during induction of anaesthesia.
2. Intravenous fluids as saline or Ringer's solution are administered to correct hypovolaemia.
3. Antibiotics: A combination of ampicillin, an aminoglycoside and metronidazole c cover all aerobic and anaerobic organisms. The antibiotics may be changed af getting the results of culture and sensitivity of pus.
4. An analgesic is given for the pain.

5. A Foley catheter is inserted to check the urine output and the adequacy of fluid replacement.

Operation

Once the general condition of the patient is satisfactory, an exploratory laparotomy is performed.

- General anaesthesia.
- Long longitudinal (midline or paramedian) incision. Pus is sampled for culture and sensitivity and is aspirated.
- The primary lesion is dealt with to stop further contamination of the peritoneum, e.g., a perforated appendix is removed and a perforated peptic ulcer is plugged by omentum.
- Peritoneal toilet by copious irrigation with sterile saline is very useful.
- Peritoneal drainage is done by suction drains coming out through separate stabs. Drainage of the subcutaneous space is also indicated as the wound is very likely to get infected.

Localized septic peritonitis (Intraperitoneal abscess)

An intraperitoneal abscess has better outcome than generalized peritonitis. It indicates that the defensive mechanisms successfully localized the source of infection. These protective mechanisms include the omentum (abdominal policeman) and the matting of loops of bowel around the source of infection.

The common sites of Intraperitoneal abscesses are the iliac fossae, pelvis and the subdiaphragmatic space (Fig. 35.3).

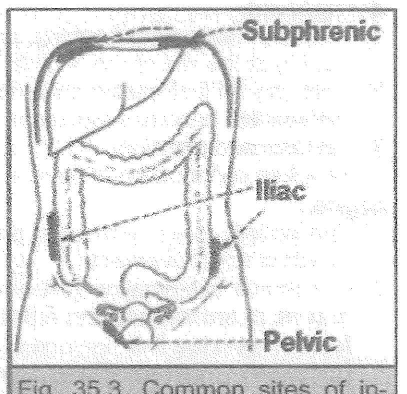


Fig. 35.3. Common sites of intraperitoneal abscesses.

Iliac abscess

Aetiology

On the right side it is due to

1. Acute appendicitis.
2. Perforated duodenal ulcer, the exudate trickling through the right paracolic gutter (Fig. 30.27).

On the left side it may be due to

1. Perforated diverticulitis.
2. Perforation of carcinoma of the sigmoid colon.

On either side

1. Spread from the female genital organs.
2. Secondary to generalized peritonitis.

Clinical features

- **Symptoms**. Pain, swelling, hectic fever, vomiting and constipation.
- Signs. Tenderness and rigidity over the site of the abscess. The overlying skin may show signs of inflammation.

Investigations

- Blood picture reveals polymorphnuclear leucocytosis.
- Ultrasound examination can determine the site of the abscess and the volume of pus.

Treatment

The principles of treatment are

1. Drainage of pus
2. Controlling the cause
3. The use of effective antibiotics.

The abscess should be drained through an extraperitoneal muscle cuffing incision. Nowadays, it is possible to do percutaneous drainage of the abscess guided by ultrasound or CT scan. An inflamed appendix is not to be removed in the acute setting. An interval appendicectomy after 6 months is much easier and safer.

Pelvic abscess

Definition

A pelvic abscess is a collection of pus in the recto-vesical pouch or in the pouch of Douglas.

Causes

1. Acute appendicitis.
2. Localization of resolving diffuse peritonitis.
3. Pelvic inflammatory disease in females.

Clinical features

1. Hectic fever and toxaemia.
2. Deep pelvic pain.
3. Diarrhoea and tenesmus are due to rectal irritation. Mucous diarrhoea occurring in a patient with an inflammatory peritoneal lesion is nearly pathognomonic of a pelvic abscess.
4. Burning micturition and frequency are due to bladder irritation.
5. Pelvic abscess may present suprapubically (mass, redness).
6. By digital rectal examination there is fullness and yielding tenderness in front of rectum.
7. If neglected, it bursts through the rectum or the vagina.

Treatment

- If the abscess is pointing in rectum, transrectal drainage is recommended.
- If it is pointing in vagina the abscess is to be drained through the posterior fornix.
- If it is pointing suprapubically, suprapubic extraperitoneal drainage is considered as a possible route. In general, provided the abscess is shut off from the general peritoneal cavity, rectal drainage of a pelvic abscess is preferable than suprapubic drainage, which breaks down nature's barriers and exposes the general peritoneal cavity to the dangers of spreading infection.

Subphrenic abscess

Anatomy of subphrenic spaces

The subphrenic region is considered as that portion of the abdominal cavity which extends from the diaphragm above to the transverse colon and mesocolon below. The region is divided by the liver into suprahepatic and infrahepatic compartments. The falciform ligament divides the suprahepatic compartments into right and left portions. The subphrenic spaces, therefore, include the following:

1. Right suprahepatic space. This is the space that lies between the right leaf of the diaphragm and the superior and anterior surfaces of the right lobe of the liver. Medially there is the falciform ligament.
2. Right infrahepatic space. (Hepatorenal Pouch of Morison). Above and in front there are the liver and gallbladder while below and behind there are the upper pole of the right kidney, lower part of the right suprarenal gland and the second portion of the duodenum (Fig. 35.4).

3. Right extraperitoneal space. The space lies between the bare area of the liver and the diaphragm.
4. Left suprahepatic space. The space is bound by the diaphragm, the left lobe of the liver, stomach and spleen.
5. Left anterior infrahepatic space. The boundaries of this space are the liver above and in front, and the stomach and lesser omentum below and behind.
6. Left posterior infrahepatic space. This is the lesser sac.
7. Left extraperitoneal space. This is the space around the upper pole of the left kidney and left suprarenal gland.

Fig. 35.4. Abscess in hepatorenal pouch of Morison.

Aetiology

1. Residual pus collection following generalized peritonitis.
2. Inflamed or perforated viscera, e.g., appendix, gall bladder or peptic ulcer.
3. Lymphatic spread from chest infection.
4. Post-operative collection of infected bile after biliary surgery.



Clinical features

A subphrenic abscess should be suspected whenever a hectic fever develops or persists after the treatment of any inflammatory lesion within the abdomen as stated, **"pus somewhere, pus no where else, pus under the diaphragm".**

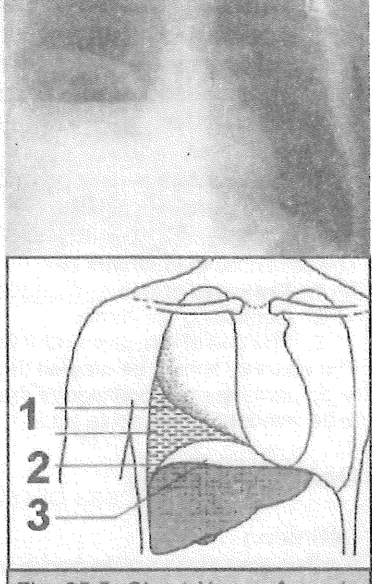


Fig. 35.5. Chest X-ray of a subphrenic abscess:
1. Rt. pleural effusion.
2. Elevated Rt hemidiaphragm.
3. Gas under diaphragm (or gas and air).

(A) General

1. Slight or absent pain but there is epigastric discomfort and pain referred to the shoulder (phrenic nerve irritation).
2. Hectic fever.
3. Tachycardia.
4. Severe toxaemia with anorexia, vomiting, sweating, wasting with rapid deterioration of the general condition.
5. Persistent hiccough.

(B) Local

Inspection

- Impaired chest movement on the affected side.
- Rarely bulging of the lower ribs or upper abdomen.

Palpation

- Tenderness may be present over the lower ribs and intercostal spaces, or below the costal margin.
- There may be swelling and rigidity of the upper abdomen.
- Downward displacement of liver with upward displacement of the apex of the heart.

Auscultation

Impaired air entry and crepitations over the lung base of the affected side may be detected.

Investigations

1. White blood cell count shows leucocytosis and shift to the left.
2. Plain chest X-ray and screen (Fig. 35.5):
   a. Thickened, elevated and fixed diaphragm (tented diaphragm).
   b. Obliteration of costophrenic space by a minimal pleural effusion may be seen.
   c. Gas under the diaphragm or air fluid level is sometimes seen when the cause is a perforated viscus, or when there is infection with gas forming organisms.
3. Ultrasound and CT scanning have proved very useful. They can localize the exact anatomical site and size of the abscess.

Treatment

If conservative treatment fails, the abscess should be drained.

A subphrenic abscess can be aspirated under ultrasound or CT guidance. A fine catheter is left in the abscess cavity to continue the drainage. Thick pus and a multilocular abscess are indications to abandon this technique in favour of open drainage by:

1. The posterior extrapleural extraperitoneal approach (when located posteriorly).
   a. The 12^{th} rib is excised subperiosteally and a transverse incision is done in its bed in line with the first lumbar transverse process through the lowest fibres of the diaphragm.
   b. A finger is inserted and worked until the abscess bursts (above the kidney between the diaphragm and liver). Fibrous septa are broken to open all loculi.
   c. A drain is inserted.
2. The anterior extraperitoneal approach (when located anteriorly).
   a. Small subcostal incision.
   b. All layers are divided but not including the peritoneum.
   c. A finger is inserted and worked until the abscess bursts, and a drain is left in place.

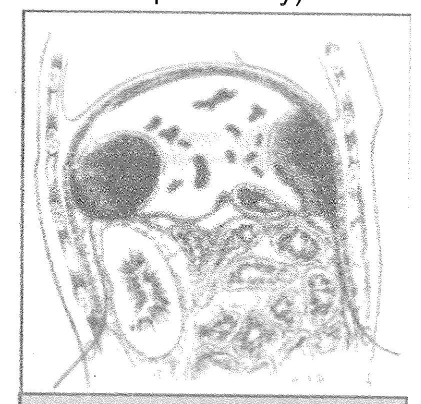


Fig. 35.6. Extraperitoneal drainage of anterior and posterior subphrenic abscesses.

Tuberculous peritonitis

Aetiology

The disease is always secondary to a tuberculous focus elsewhere that reaches the peritoneum through:

- Direct spread, e.g., tuberculous lymphadenitis, enteritis and salpingitis (the ommonest cause).
- Blood spread, e.g., pulmonary tuberculosis.
- Lymphatic spread, e.g., from pleura or bowel.

Pathology

Five types are encountered

1. The **acute miliary** type
   a. The peritoneum is studded with tubercles.
   b. The exudate is straw coloured.
   c. It resembles acute peritonitis and the diagnosis is frequently made during operation.

2. **Caseous type** (purulent form)
   a. It affects young females as a complication of tuberculous salpingitis.
   b. The peritoneum is studded with tubercles and is thickened.
   c. Multiple collections of caseous material are present among the adherent bowel and omentum. Cold abscess may form and it may point to produce sinuses or fistulae.
3. **Ascitic type** (the commonest type)
   a. The peritoneum is studded with tubercles.
   b. There is a copious amount of straw coloured fluid.
   c. The greater omentum is thickened, fibrosed and frequently rolled up forming a sausage shaped mass above the umbilicus.
4. **Encysted type** (localized form of the ascitic type)
   a. The fluid is encysted by fibrous adhesions and coils of bowel.
   b. It produces an intra-abdominal cyst which may be mistaken for ovarian and mesenteric cysts.
5. **Adhesive type** (fibrous or plastic form): This type is characterized by extensive peritoneal adhesions which may lead to intestinal obstruction.

Clinical features

1. The disease usually affects children and young adults of both sexes.
2. There are recurrent attacks of abdominal pain, vomiting and distension.
3. Tuberculous toxaemia with night fever, night sweats, anorexia and wasting is present.
4. The abdomen is felt doughy with multiple palpable swellings which may be lymph nodes, omentum and tuberculomas. Ascites is a common finding.
5. Tenderness and guarding may be present.
6. A sausage shaped mass above the umbilicus may be felt (rolled omentum).
7. Per-vaginal examination may reveal a tubo-ovarian mass.

Investigations

1. Blood picture may reveal anaemia and lymphocytosis. ESR is raised.
2. Tuberculin test is highly positive.
3. Plain chest X-ray.
4. Abdominal ultrasound may reveal loculated or free ascites or multiple swellings due to caseous collections of pus among adherent loops of intestine.
5. Abdominal tapping is performed in the ascitic type. The aspirated fluid is clear and straw coloured with a specific gravity above 1020. It is very rich in lymphocytes. It is difficult to demonstrate TB bacilli by direct films. To demonstrate them animal inoculation or culture is necessary.
6. Laparoscopy allows direct visualization of the characteristic tubercles and biopsy. A solid proof of the diagnosis is obtained, which is an essential prerequisite for the administration of anti-tuberculous drugs.
7. Exploration. In many cases a diagnosis is reached after exploration and biopsy of suspicious lesions.

Treatment

- Treatment is essentially medical. At least two antituberculous drugs, e.g. (isonicotinic acid hydrazide and rimactane) are prescribed for at least one year.
- Operation is indicated in a few cases, e.g., intestinal obstruction.

Ascites

Definition

Ascites is a pathological accumulation of fluid in the peritoneal cavity. It can be recognized clinically when the amount of fluid exceeds 1500 ml.

Aetiology

- General causes (generalized tendency for oedema formation)
  - Liver diseases, e.g. liver cirrhosis.
  - Cardiac diseases, e.g. heart failure.
  - Renal diseases, e.g. nephrotic syndrome.
  - Nutritional defects, e.g. hypoproteinaemia.
- Local causes (exudate)
  - T.B peritonitis (acute miliary, ascitic type).
  - Malignant ascites (usually secondary to carcinoma of abdominal organs).
  - Chylous ascites.
  - Pancreatic ascites.
- Rare causes
  - Meig's syndrome (due to ovarian fibroma).
  - Pseudomnoma peritonii.

Table 35.1 shows the differences between the three main causes of ascites.

Table 35.1. Differences between the main causes of ascites

	Cirrhotic ascites	Malignant ascites	Tuberculous ascites
Age	Any age	Usually elderly May be young with ovarian cancer	Usually young
History	History of jaundice or haematemesis	Short history There may be symptoms of primary tumour	Long history of low-grade fever, abdominal pain and weight loss
General examination	Features of liver insufficiency	Probably distant mets.	Low-grade fever and cachexia
Abdominal examination	Hepatomegaly and/or splenomegaly may be present	Multiple hard abdominal masses may be palpable	Doughy mass may be palpable
Liver function tests	Poor	Normal	Normal
Ultrasound	Cirrhotic liver Splenomegaly	Abdominal masses may be detected	Abdominal masses may be detected
Tapping	Transudate Specific gravity < 1020	Exudate Specific gravity >1020 Usually serosanguinous Cytology may show malignant cells	Straw coloured exudates Specific gravity >1020 Culture may reveal TB bacilli
Treatment	Medical treatment Peritoneo-venous shunt	Tapping Instillation of radioactive gold	Anti-tuberculous drugs
Prognosis	Bad	Very bad	Good

Peritoneal tumours

1. Carcinoma peritonii

Pathology

Origin

The condition is a terminal event in many cases of carcinoma of the stomach, colon, breast, ovary and other abdominal organs. It is caused by implantation of secondaries on the peritoneum.

Gross picture:

The peritoneum (both visceral and parietal) is studded with secondary nodules. The peritoneal cavity contains ascitic fluid which.is usually blood stained, but is sometimes clear and straw coloured. Peritoneal metastases may present as:

- Discrete nodules.
- Plaques variable in size and colour.
- Diffuse adhesions occur lately which may give rise to frozen pelvis.

Treatment:

Instillation of radioactive gold intraperitoneally after paracentesis.

2. Pseudornyxoma peritonii

Aetiology

- Rupture of a pseudomucinous cyst of the ovary.
- Rupture of a mucocele or mucoid carcinoma of the appendix.

Pathology

The abdomen is filled with yellow jelly-like material which is pseudomucin. A low grade inflammatory reaction occurs leading to fibrinous or fibrous adhesions.

Clinical features

Abdominal examination shows distended abdomen, without shifting dullness. Multiple firm masses are palpable.

Treatment

At laparotomy the masses of jelly-like material are scooped out and the primary focus is removed. Unfortunately the condition is liable to multiple recurrences.

3. Mesothelioma

In contrast to the above mentioned two tumours, mesothelioma is a primary neoplasm of the peritoneum, which is also known to affect the pleura. It is a rare tumour that is suspected to be caused by exposure to asbestos.

Mesothelioma is either diffuse causing ascites, or localized and bulky in which case it presents as an abdominal mass.

Torsion of the omentum

This condition is due to adhesions of the omentum to a hernia or an old focus of infection.

Clinical features

1. Sudden abdominal pain.
2. The abdomen is rigid and tender.
3. The twisted omentum may be palpable but usually the diagnosis is only made after exploration.

Treatment

Excision of the strangulated omentum.

Mesenteric cysts

A mesenteric cyst is formed of fluid collection between the 2 layers of small bowel mesentery (Fig. 35.7).



Fig. 35.7. Mesenteric cyst.

Types

1. False mesenteric cysts (without epithelial lining).
   a. Blood cyst: This is a haematoma of mesentery after trauma. Treatment is by evacuation.
   b. Tuberculous mesentric cold abscess is due to caseating tuberculous mesenteric adenitis. Treatment is by anti-tuberculous drugs.
2. True mesenteric cysts (with epithelial lining).
   a. Chylolymphatic cyst is the commonest. It is a retention cyst due to obstructed lymphatic drainage. It is thin walled, lined with endothelium and contains lymph (chyle). Its blood supply is separate from that of the related loop of intestine. Treatment: Enucleation leaving the related intestine intact.
   b. Enterogenous cyst arises from sequestrated intestinal epithelium or from duplicated intestine. It is thick walled, lined with intestinal mucosa and contains mucous. Its blood supply is derived from the same vessels of the related loop of intestine. Treatment is by excision with resection anastomosis of the related loop of intestine.
   c. Treatomatous dermoid cyst. Treatment is by enucheation.
   d. Hydatid cyst. Treatment is by enucleation.

Clinical features

Symptoms

Mesenteric cysts are encountered more in children and young adults. The patient usually complains of an abdominal swelling or dyspepsia. Pain is usually absent.

Signs

Tillaux triad:

- The cyst is near the umbilicus.
- It moves across but not along the root of mesentery (Fig. 35.8).
- The cyst is dull on percussion but there is a zone of resonance around it and a band of resonance over it due to the presence loops of bowel surrounding and in front of it.

Large abdominal cysts

The following cysts may attain a huge size

Pancreatic pseudocyst
upper abdomen

Mesenteric cyst
mid abdomen
characteristic mobility

Ovarian cyst
pelvi-abdominal

Hydronephrosis
extends to renal angle

Mesenteric lymphadenitis

Acute non-specific mesenteric adenitis

This is one of the commonest causes of acute abdominal pain in children.

Aetiology

The exact cause is unknown but may be of viral origin following respiratory infection.

Pathology

The disease involves primarily the ileocaecal lymph nodes which become discrete, soft and pink but later become firm and white. A small amount of clear serous fluid is present within the peritoneal cavity. Microscopically the nodes show a pattern of reactive hyperplasia. Culture is usually negative.

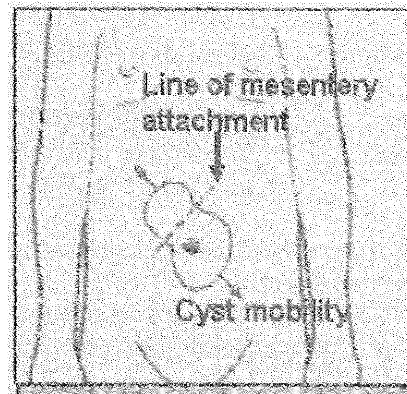


Fig. 35.8. Characteristic mobility of a mesenteric cyst perpendicular to the line of mesenteric attachment.

Clinical features

Symptoms

- It affects children and is unusual after puberty.
- It presents by an upper abdominal pain which localizes to the right side.
- The pain is colicky in nature and is severe, but it lasts for a short time. There may be nausea, vomiting, malaise, anorexia, and fever.

On examination

- Voluntary abdominal guarding but no rigidity.

Table 35.2. Differences between acute appendicitis and non- specific mesenteric lymphadenitis

	Acute appendicitis	Non-specific mesenteric lymphadenitis
Course of pain	Continuous	Periods of complete freedom of the pain
Point of maximum tenderness	McBmey's point	Above and medial to the McBurmey's point
Rovsing's sign	Positive	Negative
Shifting tenderness	Absent	Present

- Abdominal tenderness which is marked along the line of mesentery and there may be rebound tenderness.
- On rectal examination tenderness may be elicited.
- Shifting tenderness: This is a valuable sign for differentiation of the condition from appendicitis. After laying the patient on the left side for a few minutes the point of maximum tenderness shifts towards the middle line.

The clinical features are dose to those of acute appendicitis. Table 35.2 shows the clinical differences between the two diseases. Nevertheless, it is not easy to differentiate the two conditions and whenever in doubt it is much safer to operate on the patient than to miss an inflamed appendix in a child.

Treatment

Usually these patients are operated upon with a provisional diagnosis of acute appendicitis. The mesentery is found to be congested and contains multiple, soft lymph nodes. Appendectomy is performed and the post-operative course is usually uneventful.

Tabes mesenterica (Tuberculous mesenteric adenitis):

This is a childhood type of intestinal tuberculosis, that is comparable to the primary complex of the lung, but is less common. It is due to ingestion of contaminated milk. It consists of:

1. A small focus in distal ileum (Peyer's patches) comparable to Ghon's focus in the midzone of the lung (usually not detected).
2. Huge enlargement of mesenteric nodes, comparable to enlarged mediastinal nodes.

Fate

Resolution or activation takes place, depending on body resistance:

Resolution

- Healing by fibrosis and calcification.
- Later adhesions and kinking may produce intestinal obstruction.

Activation

- Caseation produces a mesenteric cold abscess.
- Rupture in peritoneal cavity produces tuberculous peritonitis.
- Spread to thoracic duct may cause miliary tuberculosis.

Clinical features (during activity):

Symptoms

1. Tuberculous toxaemia; low grade fever, sweating, wasting, and pallor.
2. Abdominal pain with vomiting and diarrhoea.

Signs

1. Tenderness below and to the right of the umbilicus.
2. Firm irregular tender masses may be felt (nodes).
3. In acute cases there are severe tenderness, rebound tenderness and rigidity, in the right iliac fossa simulating appendicitis, or along the line of the mesentery.

Investigations

1. Leucocytic count is normal.
2. Tuberculin test. If the test is negative, tuberculosis is excluded.
3. X-ray of the abdomen may show calcified nodes as mottled shadows arranged along the line of the small intestinal mesentery.

Treatment

Antituberculous treatment is given for active cases. Calcified uncomplicated nodes need no treatment.

The retroperitoneum

The retroperitoneal space is that portion of the body which is bound anteriorly by the posterior peritoneum. It is bound posteriorly by the spine and the posterior abdominal wall muscles, superiorly by the 12^{th} rib and attachments of the diaphragm and inferiorly it extends to the level of the pelvis.

There are no anatomic barriers in this area; hence pathological processes may extend easily through it.

Retroperitoneal tumours

- **Renal and adrenal tumours** are discussed in corresponding chapters.
- **Retroperitoneal sarcoma:** This commonly presents by an abdominal mass which is fixed and usually hard in consistency. The patient may complain of vague abdominal pain. The size of the lesion out-matches the symptoms caused by it. If the lesion infiltrates the colon, the patient may complain of chronic large bowel obstruction. If the ureter is obstructed by the tumour, the patient may complain of renal pain.
- Ultrasound and CT scan are very useful for the diagnosis. CT scan accurately localizes the site of the lesion, its size and reveals infiltration of adjacent structures.

Treatment

The patient is explored for the possibility of resection. Unfortunately most of the cases are unresectable and all what can be done is to take a biopsy. Radiotherapy may offer some palliation.

- **Retroperitoneal lipoma**: This lesion is more common in females, it grows slowly and it may attain a large size. It is liable to myxoematous and malignant transformation.
- **Retroperitoneal lymph node** enlargement is most commonly due to lymphomas.

THE SMALL AND LARGE INTESTINES

Surgical Anatomy

Development

The gut is embryologically formed of three parts, the foregut, the midgut and the hindgut (Table 36.1).

CHAPTER CONTENTS

Midgut rotation (Fig. 36.1)

- The yolk sac is connected to the midgut by the vitello-intestinal duct.
- In the fourth week of intrauterine life, the midgut extrudes into the umbilical cord.
- Later on, reduction of this loop with rotation happens through three stages.
  - First stage. 90° anticlockwise rotation of the midgut ends with the base of the loop lying transversely, where the small intestine rests to the right and the colon to the left.
  - Second stage. Further 180° anticlockwise rotation and reduction ends with the small intestine in the left side of the abdomen and the caecum below the liver.
  - Third stage. Descent and fixation of the caecum and proximal colon to the posterior abdominal wall.

Table 36.1. Embryological parts of gut

	Extent	Artery	Visceral pain
Foregut	Stomach and duodenum down to the ampulla of Vater	Celiac	Epigastric
Midgut	From the middle of 2nd part of duodenum (ampulla of Vater) till the junction of middle third with the left third of transverse colon	Superior mesenteric	Around umbilicus
Hindgut	Remaining part of colon, rectum, and upper half of anal canal	Inferior mesenteric	Hypogastric

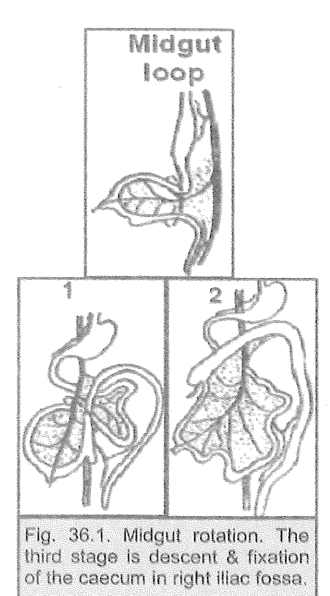


Fig. 36.1. Midgut rotation. The third stage is descent & fixation of the caecum in right iliac fossa.

Small intestine

Parts

- The small intestine is the length of bowel that extends from the pylorus to the caecum. Its length is proportionate to the body size, and is about 3 metres. The duodenum, which is retroperitoneal, ends at the ligament of Treitz. The remaining part of the small intestine is made up of the jejunum (proximal 40%), and the ileum (distal 60%).

- The jejunum is larger in diameter, thicker walled, has more prominent mucosal folds, and has less mesenteric fat than the ileum.
- The root of the small bowel mesentery extends from the left side of the body of L2 to the right sacroiliac joint. The mesentery contains fat, blood vessels, lymphatics, lymph nodes, and nerves.

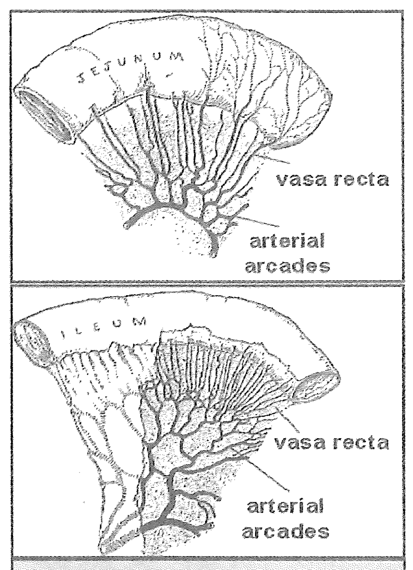


Fig. 36.2. Pattern of blood supply to jejunum and ileum.

Blood Supply
- The superior mesenteric artery supplies the jejunum and ileum by a series of straight arteries (vasa recta) which originate from arterial arcades in the mesentery.
- Venous blood drains to the superior mesenteric vein and from it to the portal vein.
- The jejunal mesentery has one or two vascular arcades, and long vasa recta; while for the ileum there are multiple arcades that extend closer to the bowel, and hence shorter vasa recta (Fig. 36.2).

The wall layers (Fig. 36.3)
- The mucosa consists of a single layer of columnar cells interspersed with mucous goblet cells, Paneth cells, and Kulchitzky cells (part of the APUD system). The absorptive surface area of the mucosa approaches 500 m^2 which is achieved by the presence of mucosal folds, villi, and microvilli on the cell surface. Under the epithelium is a thin muscle layer; the muscularis mucosae.
- The submucosa is the strongest layer and provides strength to an intestinal anastomosis. It contains lymphoid aggregates (Peyer's patches), which become more prominent distally in the ileum. The Meissner's nerve plexus is present in the submucosa.
- The muscularis propriars composed of an outer longitudinal layer, and an inner circular layer with Auerbach's myenteric plexus of ganglion cells inbetween.
- The serosa is part of the visceral peritoneum.

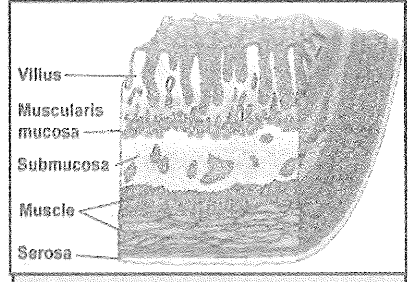


Fig. 36.3. Layers of small intestinal wall.

Colon:

Parts (Fig. 36.4)
- The colon is formed of the caecum, ascending colon, transverse colon, descending colon and sigmoid colon.
- The ileocaecal valve is a one way gate that prevents the return of colon content into the ileum.
- The caecum is a blind pouch in the right iliac fossa, and the appendix opens in its posteromedial aspect at the junction of the taeniae coli. The transverse colon joins the ascending colon at the hepatic (right) flexure, and joins the descending colon at the splenic (left) flexure, which is suspended from above by the phrenocolic ligament. The descending colon continues as the sigmoid (pelvic) colon which ends at the level of S3, where the rectum starts.

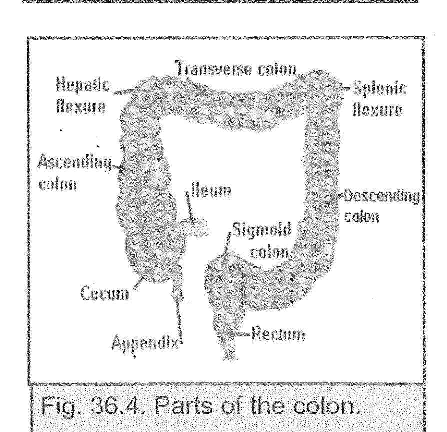


Fig. 36.4. Parts of the colon.

- The colon is characterized by the taeniae coli and the appendices epiploicae which are multiple, small, pedunculated fat pads on its outer surface. The rectum has neither taeniae nor appendices epiploicae.
- The caecum is covered by peritoneum on both sides and on the anterior surface. It is also commonly covered by peritoneum posteriorly forming a retrocaecal space in which the appendix frequently lies. The transverse colon and the sigmoid colon are completely covered by peritoneum, have mesenteries, and are mobile. The sigmoid mesocolon has an inverted *"V'* attachment to the pelvic brim, the apex of the "V' overlies the left ureter as it cross the bifurcation of the left common iliac artery. The greater omentum is attached to the antimesenteric border of the transverse colon.

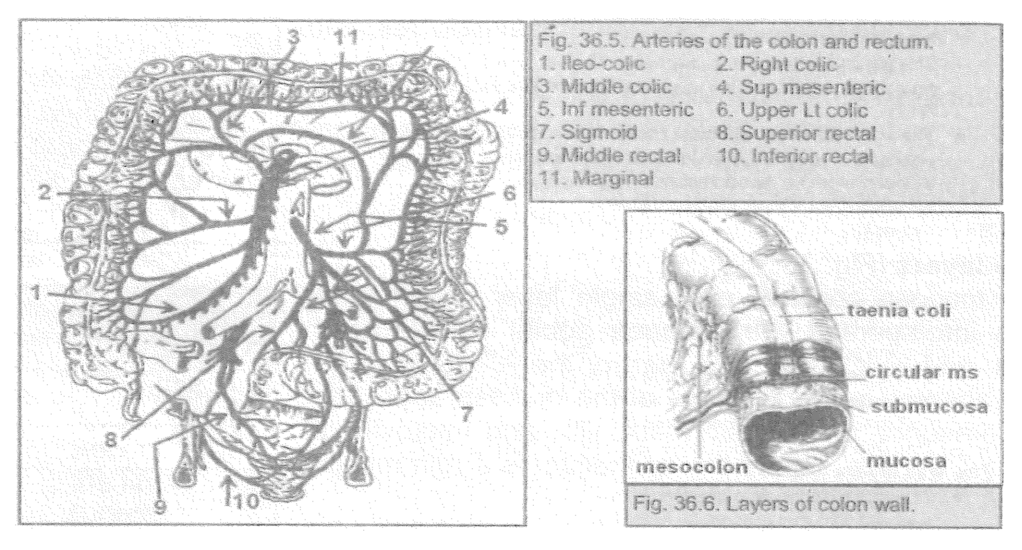


Fig. 36.5. Arteries of the colon and rectum.
1. Ileo-colic 2. Right colic
3. Middle colic 4. Sup mesenteric
5. Inf mesenteric 6. Upper Lt colic
7. Sigmoid 8. Superior rectal
9. Middle rectal 10. Inferior rectal
11. Marginal

Fig. 36.6. Layers of colon wall.

Blood supply

Arteries (Fig. 36.5)

- The superior mesenteric artery supplies the mid-gut portion of the colon by:
  - Ileocolic artery is a terminal branch of the superior mesenteric, and supplies blood to the appendix via the appendicular artery, and to the caecum and proximal ascending colon.
  - Right colic artery supplies the remainder of the ascending colon and the hepatic flexure.
  - Middle colic artery supplies the transverse colon except for its left third.
- The inferior mesenteric artery supplies the hindgut portion of the colon by:
  - Left colic artery supplies the left third of the transverse colon, splenic flexure, and descending colon.
  - Sigmoid branches to the sigmoid colon.
- The marginal artery of Drummond connects all the mentioned arteries as it lies in the concavity of the colon. It is an important collateral channel between the superior and inferior mesenteric arteries. This marginal artery has a great surgical importance as it can maintain the viability of a long segment of the colon after division of a major colic branch. This allows colon bypass operation to be feasible.

Veins. The veins parallel the arteries similarly named, and utimately drain in the portal vein.

Lymphatic drainage

Lymph drains from the colon to the following 1ymph node groups, in sequence;

1. Epicolic nodes on the bowel wall.

2. Paracolic nodes between the marginal artery and the bowel.
3. Intermediate nodes on the main vessels.
4. Principal nodes alongside the superior and inferior mesenteric vessels.

Nerve supply

- The parasympathetic nerve supply to the right and transverse colon is through the vagus nerve, while the distal colon and the rectum are supplied by the nervi erigentes (the pelvic splanchnic nerves) which originate from S2,3,4. The parasympathetic system is motor to the bowel wall.
- The sympathetic system supplies the blood vessels of the large intestine through the greater and lesser splanchnic nerves.

The wall layers (Fig. 36.6)

- The mucosa of the large intestine is formed of columnar epithelium and numerous mucus secreting goblet cells. Under the epithelium is a thin muscle layer; the muscularis mucosae.
- The submucosa is formed of connective tissue and is the strongest layer of the bowel wall.
- Muscularis propria is formed of an inner circular layer, and an outer longitudinal one that is condensed into three bands (taeniae coli).
- The serosa is part of the visceral peritoneum.

Rectum

Features and relations

- The rectum, which is 12-15cm in length, starts at the recto-sigmoid junction opposite the third sacral segment, passes downwards in the hollow of the sacrum, to end at the anorectal junction at the level of the pelvic floor.
- The rectal ampulla is the distal part of the rectum, which functions as a reservoir because of its characteristic distensibility.
- The rectum is not a straight tube as its name implies. It describes three lateral curves, the inner aspects of which form the "valves of Houston" that are seen on endoscopic examination (Fig. 39.1).
- The rectum has neither taeniae coli, haustrations, nor a mesentery, and is partially covered by peritoneum. The upper third of the rectum is covered on the front and both sides by peritoneum, the middle third is peritonealized only anteriorly, while the lower third is completely devoid of peritoneal covering.
- Posteriorly, the rectum is loosely bound to the sacrum by a condensation of connective tissue (the fascia of Waldeyer), behind which lies the pelvic plexus of autonomic nerves. Anteriorly, the tough fascia of Denonvillier separates it from the back of the urinary bladder, and the prostate in males. Laterally the rectum is attached to the side walls of the pelvis by lateral ligaments which contain the middle rectal arteries. The fascial layers in front of and behind the rectum act as barriers against malignant permeation.

Arterial supply: (Fig. 36.5)

- The superior rectal artery is the direct continuation of the inferior mesenteric artery and is the main blood supply to the rectum.
- Right and left middle rectal arteries arise from the internal iliac arteries and run medially in the lateral rectal ligaments.
- Right and left inferior rectal arteries arise from the pudendal arteries and cross the ischiorectal fossa to reach the anal canal.

Venous drainage

- The veins parallel the arteries and are similarly named. The superior rectal vein becomes the inferior mesenteric vein which ultimately drains via the portal circulation.
- The middle and inferior rectal veins drain via the systemic circulation.
- The region of the lower rectum and anal canal represents one of the sites of porto-systemic collaterals. Patients with portal hypertension may develop rectal varices.

Lymphatic drainage

- The main lymphatic drainage is in an upward direction to lymph nodes along the superior rectal vessels, then to the inferior mesenteric lymph nodes and finally to the pre-aortic lymph nodes.
- The lower part of the rectum may drain in addition to the middle rectal lymph nodes along the lateral ligaments to the internal iliac lymph nodes.

Surgical physiology

Functions of the small intestine

- The main function of the small bowel is absorption. Fat, protein, carbohydrates, vitamins, water, and electrolytes are all absorbed.
    - With a few exceptions (e.g. iron and calcium), the small intestine absorbs indiscriminately without regard to body composition.
    - To be absorbed, vitamin B_{12} combines with the intrinsic factor secreted by the stomach. It is then absorbed in the distal ileum. Resection of a long segment of the distal small intestine is, therefore, likely to produce vitamin deficiency.
- The small intestine has also important secretory and digestive functions through the succus entericus. Ingested fluid and salivary, gastric, biliary, pancreatic, and intestinal secretions present a total of 5-9 L/day, but only 1-2 L pass the ileocaecal valve to the colon.

Functions of the colon and rectum

1. Absorption of water and sodium to concentrate the waste products. This is mainly a function of the right side of the colon.
2. The left side of the colon acts as a reservoir for solid faeces until the time of defaecation.
3. Mucus secreted by the mucosa acts as a lubricant. Potassium and bicarbonate are also secreted in small quantities. Excessive diarrhoea may result in potassium and bicarbonate loss and metabolic acidosis.

Principles of colon surgery

Colon surgery deserves special consideration because compared to the small bowel; its anastomosis is more liable to disruption, leakage and peritonitis. The factors of difference are:

1. The highly infective content of both aerobic and anaerobic organisms.
2. Constant gaseous distension.
3. Incomplete serous coat.
4. Peculiar blood supply where the terminal arteries poorly connect with each other.

Preoperative preparation for elective surgery

Patient counseling particularly with those who are likely to have an intestinal stoma.

Improving nutritional status.

Bowel preparation

The risk of anastomosis leakage and wound sepsis is reduced, if the large bowel is empty at the time of resection and if the bacterial flora of the colon is reduced. In elective cases where the colon is well prepared, which means that it is empty and clean, primary anastomosis (i.e. in the same session as the resection) can be performed. There are two methods of bowel preparation.

Mechanical preparation

- Standard preparation
  - Non residue diet for 4 days before surgery.
  - Enemas and mild laxatives for 2-3 days before the operation.
- Rapid preparation is an alternative that can be performed one day before surgery by one of the following methods.
  - Whole gut irrigation using 2-4L/hour of a balanced crystalloid solution passed via a nasogastric tube until the patient passes clear fluid per rectum. This method is not used in patients with known cardiac, or renal disease, and in those with partially obstructed colon.
  - Mannitol. One litre of flavoured mannitol is given orally or by a naso-gastric tube. Metchlopramide may be administered to inhibit vomiting.
- **Chemical preparation**. Intestinal antiseptics administered orally help to reduce the density of colon bacteria. A combination of neomycin and metronidazole (Flagyl) for two days will cover the Gram negative bacilli and the anaerobes which are normally resident in the large intestine.

Prophylactic parenteral antibiotics

To minimize septic complications following colorectal surgery, systemic antibiotics are administered immediately before surgery and are continued postoperatively for one day. This is termed "perioperative antibiotic prophylaxis" (chapter 7), and is usually a combination of a cephalosporin or an aminoglycoside with either metronidazole or clindamycin.

Operative procedure

Resection

- The extent of resection is govemed by the arterial blood supply and by the disease process.
- In radical surgery for malignant tumours, it is essential to remove the draining lymphatics.
- Division of the peritoneal attachments allows adequate mobilization of the bowel on its mesentery.

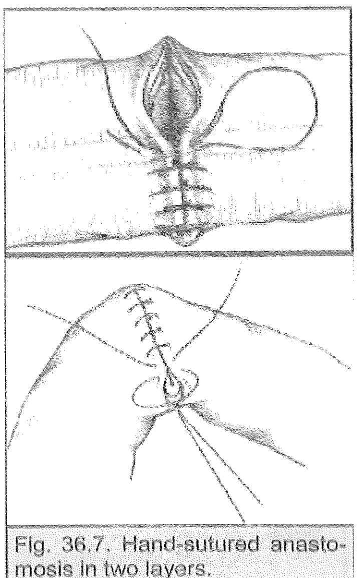


Fig. 36.7. Hand-sutured anastomosis in two layers.

Anastomosis

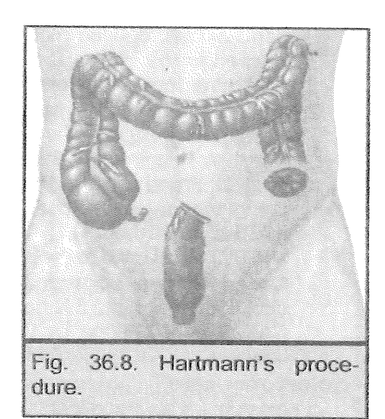
For a successful anastomosis the two bowel ends should be adequately vascularized, and should be sutured without tension. An intestinal anastomosis can be done by one of two methods.

1. Hand suturing is commonly done in two layers of interrupted sutures. The first layer includes the whole wall thickness, while the second incorporates the serosa and muscle only, to invert and seal the suture line (Fig. 36.7). Some surgeons prefer a single layer of interrupted sutures. The defect in the mesentery is closed to prevent an internal hernia.
2. Mechanical staplers are increasingly used. They are faster but more costly.

Emergency surgery

In patients with obstruction, perforation, toxic dilatation, or massive bleeding from the colon, emergency surgery may be required.

- In critically ill obstructed cases, a temporary proximal colostomy to decompress the colon is done, postponing resection to a later date.
- If the patient's condition allows, resection of the diseased colon should be carried out in the course of the emergency operation. After excision of the diseased segment, the surgeon is faced with a colon that is heavily loaded with stools and bacteria, the decision for further management depends on the site of the resected part.
    - With right colon resection, restoration of bowel continuity by primary anastomosis is feasible. The ileum is anastomosed to the transverse colon, i.e. an ileo-transverse anastomosis.
    - With emergency excision of other parts of the colon, primary anastomosis is avoided because of the high possibility of disrupted suture line and leakage. The options are:
        - The proximal colon end is opened to the skin as a temporary colostomy, and the distal end is closed by sutures and replaced in the abdomen (Hartmann's procedure, Fig. 36.8).
        - Both ends are opened to the skin, the proximal one as a temporary colostomy, and the distal one as a mucus fistula.


Fig. 36.8. Hartmann's procedure.

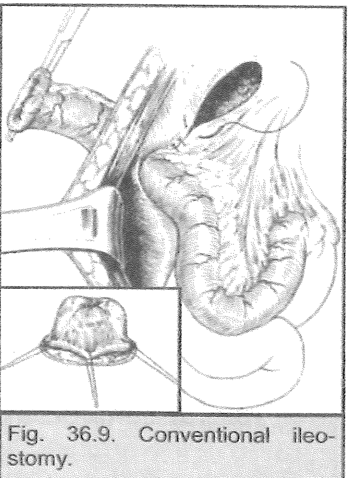
In either case a second operation is needed to restore bowel continuity within a few weeks. This second elective operation should be preceded by proper bowel preparation.

Intestinal stomas

Ileostomy

Indications

Proctocolectomy for ulcerative colitis or familial polyposis coli.


Fig. 36.9. Conventional ileostomy.

Conventional ileostomy (Fig. 36.9)

The stoma is fashioned so that a nipple of ileum protrudes from the skin, facilitating direct delivery of the irritant small bowel content into an appliance. Conventional ileostornosis are incontinent.

Continent ileostomy

A continent ileostomy may be done by fashioning a valve with an underlying reservoir (Kock pouch) which the patient regularly evacuates by passing a tube.

Colostomy

A colostomy is an opening of the colon to the skin.

Indications

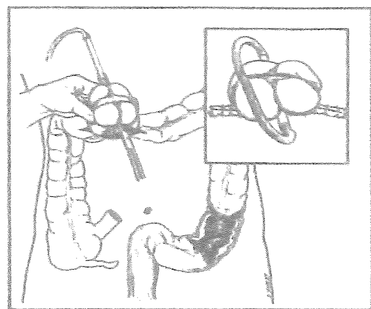
Temporary colostomy

1. To relieve large bowel obstruction in patients with:

a. High anorectal malformations.
b. Hirschsprung's disease.
c. Inflammatory stricture.
d. Carcinoma of the colon.

After the obstruction is relieved, the colon is prepared and the cause is corrected. Later, the colostomy is closed. This is called three-stage management of acute colon obstruction.

2. Injuries of the colon. The injured segment is either exteriorized as a colostomy, or is closed and a proximal diverting colostomy is performed.
3. To protect a distal doubtful colonic or rectal anastomosis.



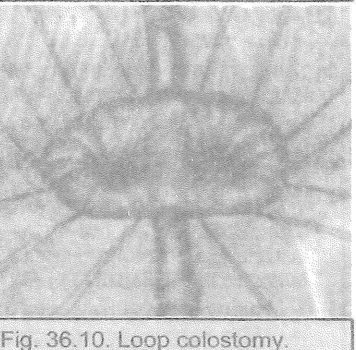


Fig. 36.10. Loop colostomy.

Permanent colostomy

1. After abdomino-perineal resection.
2. Irresectable carcinoma of the large bowel with obstruction.
3. Incurable cases of anal incontinence.
4. High anal fistulae that is not amenable to surgery.

Types

1. Loop colostomy (Fig. 36.10) is usually temporary and is usually done in the right side of the transverse colon (transverse colostomy). This part is brought to the surface, fixed to the abdominal wall, opened along one of the taeniae coli, and then stitched to the skin. A rod is passed beneath the loop to prevent retraction, and is removed after one week.
2. End colostomy may be permanent or temporary. It is commonly constructed in the left or the sigmoid colon (iliac colostomy) by bringing the divided end of the bowel to the surface (Fig. 36.8 and 36.11). The lateral space between the exiting colon and the parietal peritoneum is closed to prevent an internal hernia. The colon is fixed to the abdominal wall muscles and is then stitched to the skin.

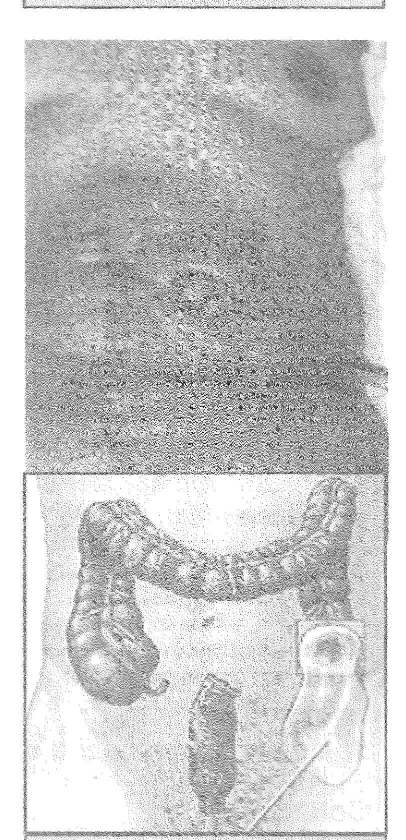


Fig. 36.11. Terminal colostomy and its appliance.

Colostomy care

A colostomy is an incontinent opening on the abdominal wall.

1. An iliac colostomy is easy to manage, as by time it functions once or twice a day. The patient usually uses a colostomy appliance and evacuates its plastic bag when it is full (Fig. 36.11). An alternative method is to wash out the colon through the colostomy once every morning. As the colostomy does not act during the rest of the day, a simple dressing is all that is necessary.
2. A transverse colostomy is wet, i.e., it discharges semi-liquid stools frequently, and, therefore, requires an appliance.

Colostomy complications

1. Prolapse.
2. Retraction.
3. Necrosis of distal end.
4. Stenosis of the orifice.
5. Colostomy hernia. This is either external coming out through the anterior abdominal all opening beside the colon end, or internal when the surgeon fails to close the space lateral to the exiting colon between it and the parietal peritoneum.

Intestinal trauma

The small and large intestines are frequently injured, either alone or in combination with other intra-abdominal viscera. The prognosis after treatment of intestinal injury largely depends on these associated injuries. Colon injuries are more dangerous than those of the small intestine because of the magnitude of intra-abdominal contamination.

Aetiology

The intestine may be injured by:

1. Blunt abdominal trauma as in road traffic accidents (RTA) or blows to the abdominal wall. Presence of seat belt injuries should direct attention to the possibility of intestinal injury.
2. Penetrating trauma as stabs and bullets. High velocity missiles are more damaging than low velocity ones and stabs. Indirect trauma, e.g., blast injuries are known to affect the colon.
3. latrogenic injury is increasing with the rising popularity of invasive investigations. Examples include:
   a. The duodenum may be injured during an attempt at endoscopic sphincterotorny that is done to remove a stone from the common bile duct.
   b. The colon may also be injured with colonoscopic diathermy excision of polyps.
   c. The intestine may be injured during laparoscopic surgery when the pneumoperitoneum needle is introduced in an area where the intestine is adherent to the anterior abdominal wall.

Types of injury

1. Contusion and haematoma.
2. Rupture which may be complete or incomplete and single or multiple.
3. Tears of mesentery or mesenteric vessels resulting into haematomas, and gangrene of the affected bowel segments.

Sequelae

1. Peritonitis occurs due to the escape of intestinal contents into the peritoneum.
2. Internal haernorrhage. With intestinal injuries bleeding is less of a problem than peritonitis.
3. Hypovolaemia and septic shock.
4. Paralytic ileus.
5. Injuries may heal by strictures and massive adhesions presenting later by intestinal obstruction,

Clinical features

Symptoms

- History of trauma
- Abdominal pain which starts at the site of injury and later spreads.

Signs

- General signs. Tachycardia, fever, and hypotension.
- Local signs
    - Signs of injury in the abdominal wall, e.g., bruises or the inlet and exit wounds of missiles.
    - Tenderness and rebound tenderness.
    - Lost liver dullness due to free air in the peritoneum.
    - Shifting dullness because of free intraperitoneal fluid.
    - Distension due to developing ileus.
    - It should be stressed that the frank picture of peritonitis may be delayed in patients with a tiny perforation. Sometimes, the injured segment is contused and it takes some time to perforate. Whenever in doubt, it is better to keep the patient under observation in the hospital for 24 hours.

Investigations

The diagnosis of bowel injury is mainly based on clinical grounds. Investigations are only resorted to in doubtful cases.

1. Laboratory tests. Leucocytosis, and haemodilution,
2. Radiology
    - Plain X-ray of the abdomen may show:
        - Free air under the diaphragm.
        - Multiple fluid levels due to ileus.
        - Bullets and sharpnels.
        - Fractures.
    - Ultrasound or CT scan may reveal a haematoma or intrapreitoneal collection.
3. Diagnostic peritoneal lavage (DPL) may show free blood, bile, or intestinal contents (Chapter 40).

Treatment

All intestinal injuries require laparotomy.

Priorities of multiple trauma management (Chapter 2) should be followed:

- Preoperative preparation
    - Anti-shock measures.
    - Antibiotics that cover the spectrum of Gram-negative bacilli and anaerobes should be administered, e.g., a combination of a third generation cephalosporin and metronidazole.
    - Tetanus toxoid boosting dose.
    - Insertion of a nasogastric tube and a self retaining urethral catheter.
- Operation (abdominal exploration = laparotomy)
    - Midline abdominal incision,
    - Full abdominal exploration for solid organs (liver, spleen, pancreas) and kidneys), and for hollow organs (stomach, small and large intestine) should be done.
    - Bleeding should be urgently stopped. Leaking intestine is dealt with afterwards.
    - Small intestinal and right colon injuries:
        - Tidy sharp injuries are sutured in two layers.
        - Ragged injuries require trimming of the devitalized edges (freshening of edges), and suturing.
        - Intestinal resection is indicated for ischaemic or gangrenous segments due to mesenteric vessels injury, extensively contused segments, and

for multiple tears that are very close to each other. Restoration of intestinal continuity by primary anastomosis is the rule.

- Transverse and left colon injuries
  - Localized injuries are exteriorized, i.e., the injured part is brought out to the skin through an opening in the anterior abdominal wall. It thus acts as a colostomy. If the injured part cannot be exteriorized the tear is sutured, and is protected by a proximal colostomy to divert the faeces away. In either case, the colostomy is electively closed after three weeks with adequate colon preparation.
  - In cases where resection of a segment is done, intestinal anastomosis is not attempted in the same session. The proximal end of the colon is brought out as a terminal colostomy. The distal end is either brought out as a mucus fistula, or is closed (Hartmann's procedure). Elective restoration of bowel continuity is done after three weeks.
  - Before abdominal closure, the peritoneal cavity is irrigated with copious amounts of saline.

Intestinal fistulae

Aetiology

1. Eighty percent of external fistulae develop after an abdominal operation. The cause is either:
   a. Unrecognized intestinal injury.
   b. Failure of an intestinal anastomosis. Causes of this failure include:
      i. Poor vascularity.
      ii. Anastomosis under tension.
      iii. Anastomosis in the presence of sepsis.
      iv. Distal obstruction.
      v. Lack of proper surgical technique.
      vi. Presence of a specific pathology as Crohn's disease.
      vii. Generalized diseases that impair healing, e.g., hypoproteinaemia.
2. **Other causes**
   a. Congenital anomahes as a patent vitello-intestinal duct that discharges at the umbilicus.
   b. Abdominal trauma.
   c. Inflammatory conditions as colonic diverticulitis, Crohn's disease, and radiation enteritis. These produce external or internal fistulae.
   d. Malignant tumours.

Pathology

A fistula is an abnormal communication between two epithelialized surfaces. The communication is usually formed by granulation tissue, but in some cases it may be lined by epithelium.

Classification

1. Intestinal fistulae are classified into internal when they connect the intestine to hollow viscera (another part of the gut, urinary tract, or vagina), or external when it is connected to the skin.
2. External fistulae are further classified into low-output fistulae that discharge less than 500 ml/day, and high-output fistulae that discharge more than 500 ml/day. The amount of fistula output has an important bearing on its metabolic effects.
3. According to origin in intestine. This is roughly known by the nature of discharge.

- Bile-stained discharge indicates an origin from he duodenum or jejunum.
- Fluid faecal discharge indicates an origin from the ileum or caecum.
- Semisolid faecal discharge indicates an origin from the distal colon.

Complications

1. **Metabolic effects** are particularly common with high output fistulae. These are the results of malabsorption and loss of intestinal contents. Sepsis, if present, aggravates the malnutrition as it leads the patient into severe catabolism. Metabolic effects include:
    a. Dehydration.
    b. Malnutrition, e.g., hypoalbuminaemia.
    c. Electrolyte disturbances as hypontraemia and hypokalaemia.
    d. Acid-base disturbance causes acidosis.
2. **Sepsis**. In some cases, the fistula track is not effectively walled off, and enteric contents escape producing an intraperitoneal abscess. Sepsis is the major problem in patients with intestinal fistulae.
3. **Skin irritation and maceration** due to continuous flow of intestinal contents.

Management

The great majority of intestinal fistulae heal spontaneously if sepsis is eradicated, the nutritional status is maintained and distal obstruction, if present, is relieved. Managing an intestinal fistula proceeds in steps.

1. **Resuscitation and skin protection**
    a. The priority is to save the life of the dehydrated patient by intravenous fluids to restore normal blood volume, fluid, and electrolyte balance. Blood transfusion may be needed.
    b. Skin protection should start as early as the fistula is recognized for if excoriation occurs, skin management becomes difficult. An adhesive cover and a disposable collection bag are applied to the skin around the fistula. The skin is protected and the effluent can be measured.
2. **Nutritional support** (Chapter *9)*
    a. Parenteral nutrition is instituted for patients with high output, or proximal fistulae.
    b. Patients with low output and distal fistulae can make use of their intestine for absorption. Enteral nutrition is indicated.
3. Investigations and eradication of sepsis. The aims of investigations are to detect the level of the fistula, the presence of bowel disease, the presence of distal obstruction, and the presence of intraperitoneal sepsis. The means of investigation are:
    a. Clinical assessment.
    b. Fistulography and barium meal follow-through.
    c. Ultrasound or CT scan of the abdomen for detection of abscess cavities. An abscess should be drained either surgically or is aspirated under sonographic or CT guidance.
4. **Definitive treatment**

- **External fistula:**
    - Continued conservative treatment with nutritional support is indicated in patients showing progressive diminution of the output. This is successful in most cases.
    - Surgical intervention is indicated for:
        - Cases that show no improvement over 4-6 weeks.

- Distal obstruction.
- Active disease at the fistula site, e.g. Crohn's disease or malignancy.
- Total discontinuity of the bowel ends.
- Mucocutaneous continuity.

A good nutritional status allows resection of the diseased bowel segment and anastomosis in one stage.

- **Internal fistula**. Spontaneous closure is rare to occur, and many internal fistulae do not require correction. Surgery, in the form of resection of the affected intestinal segment and anastomosis of healthy ends, is indicated for:
  - Patients with nutritional problems where a fistula bypasses a long segment of intestine producing malabsorption.
  - Internal fistulae to the urinary tract.

Intestinal diverticula

A diverticulum is a blind pouch that is continuous with the lumen of a hollow viscus (gut or urinary bladder).

Diverticula can occur anywhere in the bowel from the duodenum down to the sigmoid colon. The common ones, however, are diverticula of the colon and Meckel's diverticulum.

Meckel's diverticulum

Aetiology

A Meckel's diverticulum is the most prevalent congenital anomaly of the gastrointestinal tract. It is caused by persistent patency of the proximal part of the vitello-intestinal (omphalo-mesenteric) duct of the embryo (Fig. 36.12 and 36.13). The abnormal persistence of the duct may produce other anomalies as well:

- A faecal fistula connecting the ileum with the umbilicus, caused by persistent patency of the whole vitello-intestinal duct (Fig. 41.4).
- A fibrous band remaining after obliteration of the duct (Fig. 36.14).
- An enterocyst that results from the persistent patency of the middle part of the duct.
- A persistent umbilical extremity of the duct is everted producing an umbilical polyp (Fig. 41.5).

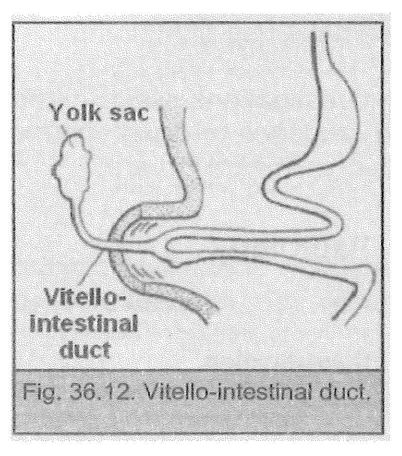


Fig. 36.12. Vitello-intestinal duct.

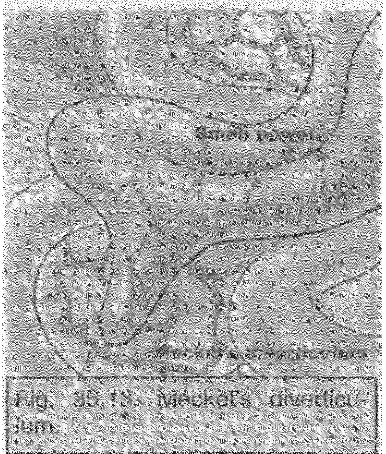


Fig. 36.13. Meckel's diverticulum.

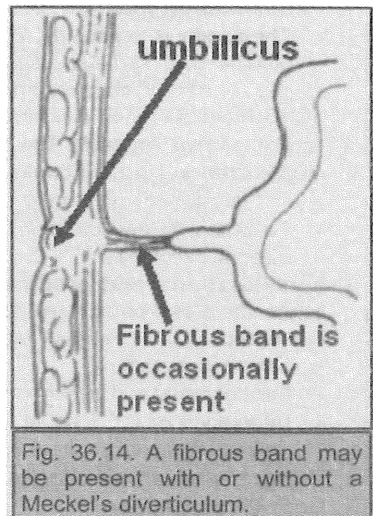


Fig. 36.14. A fibrous band may be present with or without a Meckel's diverticulum.

Pathology

Meckel's diverticulum is a true diverticulum that incorporates all layers of the bowel wall. It lies on the antimesenteric border, and has its separate blood supply via a branch of

the superior mesenteric artery. It may contain ectopic gastric mucosa, and sometimes pancreatic and colonic cells.

2-3 rule. As an approximation:
- It occurs in 2-3% of the population
- It is situated in the ileum 2-3 feet from the caecum
- It averages 2-3 inches in length.
- Symptoms are due to complications which occur in about 2-3% of affected people.

Complications
1. Intestinal obstruction is the commonest complication. It may be due to:
   - Intussusception: The ectopic mucosa at the base of the diverticulum acts as a foreign body and forms the apex of an ileo-ileal intussusception.
   - Pressure on an intestinal loop by a fibrous band that may attach the diverticulum to the umbilicus, the band may also allow rotation of the ileum around its axis (volvulus).
2. Incarceration in an inguinal or a femoral hernia is called Littre's hernia.
3. Peptic ulceration and bleeding are caused by the ectopic gastric mucosa, particularly in childhood. The ulceration occurs in the intestinal mucosa adjacent to the base of the diverticulum. Bleeding secondary to a Meckel's diverticulum is the commonest cause of lower gastrointestinal bleeding in children.
4. Acute diverticulitis is similar to acute appendicitis. Gangrene and perforation may occur and leads to peritonitis (Fig. 36.15).

Fig. 36.15. Acute Meckel's diverticulitis with gangrene.

Clinical features
1. A Meckel's diverticulum may be accidentally discovered at operation for another pathology.
2. It may present by virtue of its complications:
   - Intestinal obstruction.
   - Painless rectal bleeding in childhood.
   - Acute abdominal pain simulating appendicitis. Clinically it is impossible to differentiate the two conditions. The patient is usually explored on the provisional diagnosis of acute appendicitis. Finding a normal appendix at operation, the surgeon should look for a Mecker's diverticulum.

Treatment
Symptomatic cases. Resection is indicated.

Accidentally discovered at laparotomy
- Resection is indicated in children and young adults and in those with an attached band.
- In other instances, particularly in patients over 40 years, the potential risks of resection outweigh the advantages.

Diverticular disease of the colon
Aetiology
The aetiology of the disease is supposed to be lack of fibre in the diet leading to chronic constipation and increased intraluminal colonic pressure. Increased muscle spasm and segmentation may playa role.

Pathology

Nature. This disease represents pulsion diverticula of the colonic mucosa (Fig. 36.16) through the circular muscle layer at the points of entery of the blood vessels between the taenia coli.

Site. The sigmoid colon is the commonest site affected, but any area of the colon may be involved. The rectum is never affected.

Development. In the early stages of the disease there is only muscular spasm and incoordination. Next, the mucosa starts to bulge outwards through the circular muscular layer at the points of entery of blood vessels between the taenia at the antimesenteric border (Fig. 36.17). This stage is called non complicated diverticuar disease or diverticulosis.

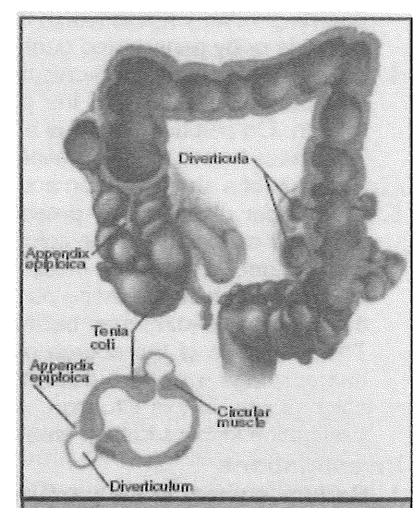


Fig. 36.16. Colon diverticula are mucosal out-pouchings that are more frequent in the sigmoid colon.

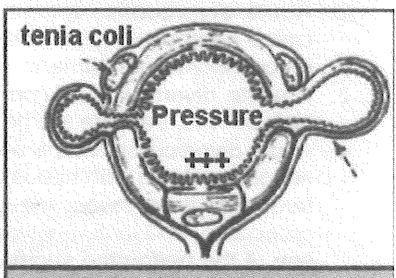


Fig. 36.17. Development of colon diverticula

Complications

1. Acute diverticulitis is inflammation of one or more of the diverlicula, usually secondary to obstruction of its neck.
2. Perforation may be a sequel of acute diverticulitis. It may lead to localized or generalized peritonitis.
3. Recurrent attacks of acute inflammation.
4. Bleeding due to erosion of a blood vessel at the neck of a diverticulum by inspissated (Fig. 36.18).
5. Colon stricture secondary to chronic diverticulitis. The result is large bowel obstruction.
6. Fistula formation. A colovesical, colovaginal or colointestinal fistula may develop.

Clinical features

1. Diverticular disease is uncommon before the age of 40 years. Afterwards its incidence increases with age.
2. In the stage of diverticulosis the patient may be asymptomatic or complains of recurrent attacks of lower abdominal pain, distension and flatulence.
3. Acute diverticulitis mimics the clinical picture of appendicitis but on the left side. There is abdominal pain, pyrexia with tenderness and guarding in the left iliac fossa. It may be complicated by a pericolic abscess or by generalized peritonitis.
4. A patient with chronic diverticulitis will give a long history of recurrent attacks of pain with the passage of blood and mucous per rectum. On palpation there is a tender mass in the left iliac region. This mass has to be differentiated from carcinoma. In the latter the history is of a short duration and pain is absent in 25% of cases.
5. Diverticular disease may present with fresh bleeding per rectum. Although carcinoma of the colon is a commoner cause of bleeding per rectum, diverticular disease is one of the two commonest causes of massive bleeding per rectum (Besides angiodysplasia of the colon). Bleeding may be the first presentation of the disease. The diagnosis of the source of bleeding in patients with profuse haemorrhage may be a difficult problem as it is difficult to perform colonoscopy in this situation. Angiography is the most useful investigation in this situation (see Chapter 40).

Investigations

1. Barium enema. In the prediverticular stage a saw tooth appearance of the colon is present. Fully developed diverticula will be visualized and their extent is known (Fig. 36.19). Barium enema can diagnose a concomitant lesion. Barium enema is contraindicated during acute diverticulitis.
2. Sigmoidoscopy will detect the mouths of the diverticula (Fig. 36.20). It is of more value to diagnose any concomitant lesion.
3. C.T scan is the best investigation in cases of acute diverticulitis. It reveals thickening of the colonic wall, a peridiverticular abscess or extraluminal gas.

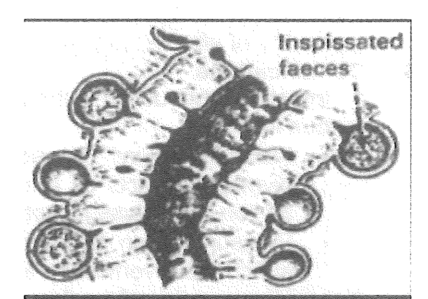
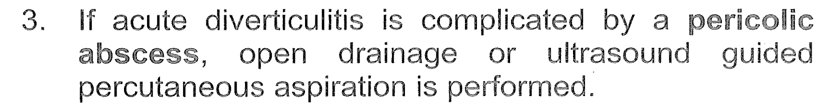


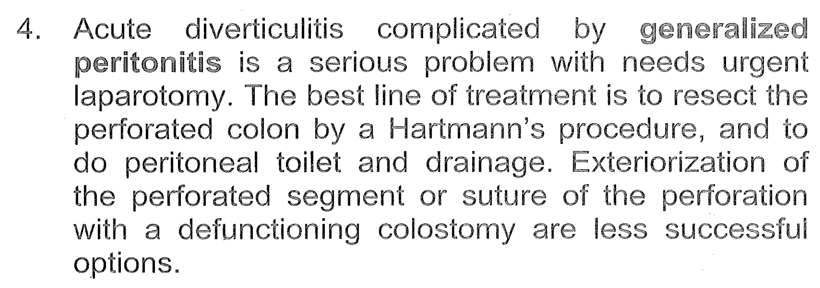
Fig. 36.18. Inspissated feaces causes erosions at the neck of a diverticulum and bleeding.

Treatment

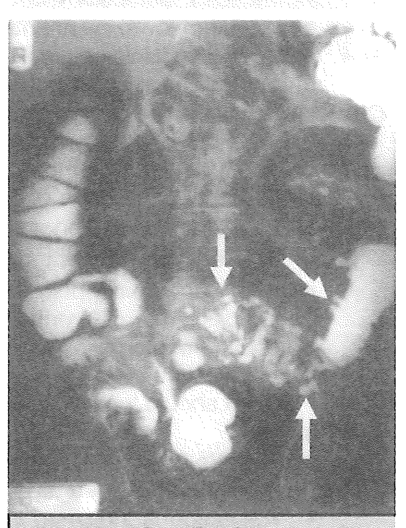
1. In the stage of diverticulosis a high fibre diet is prescribed. Antispasmodics may be helpful for the abdominal colics.
2. **Acute diverticulitis** is treated conservatively on the same principles as appendicular mass. Usually the inflammation settles on conservative treatment.



3. If acute diverticulitis is complicated by a **pericolic abscess**, open drainage or ultrasound guided percutaneous aspiration is performed.

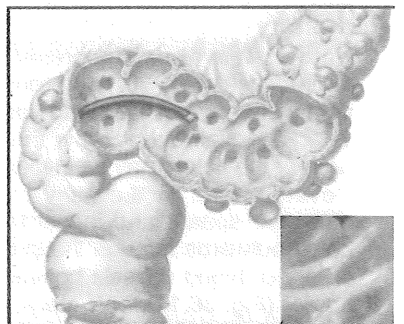


4. Acute diverticulitis complicated by **generalized peritonitis** is a serious problem with needs urgent laparotomy. The best line of treatment is to resect the perforated colon by a Hartmann's procedure, and to do peritoneal toilet and drainage. Exteriorization of the perforated segment or suture of the perforation with a defunctioning colostomy are less successful options.
5. **Chronic diverticulitis**. Colectomy is performed after adequate mechanical and chemical preparation of the colon.
6. **Bleeding** diverticular disease is treated by adequate resuscitation. Fortunately in the majority of patients, the bleeding stops spontaneously. If the bleeding is massive or persistent, angiographyis done to localize the site of bleeding and urgent colectomy is performed.


Fig. 36.19. Barium enema shows diverticula of sigmoid colon

Inflammatory bowel disease

The term "inflammatory bowel disease" is usually used to denote ulcerative colitis and Crohn's disease. However, bowel inflammation can be caused by other diseases, e.g. schistosomal colitis, amoebic colitis, ileocaecal tuberculosis, and typhoid enteritis.


Fig. 36.20. Sigmoidoscopy for colon diverticula.

Ulcerative colitis

As the name indicates, it is an inflammatory disease of the colon (colitis) characterized by ulcerations.

Aetiology is unknown. Immunologic, genetic, and environmental factors are possible causes.

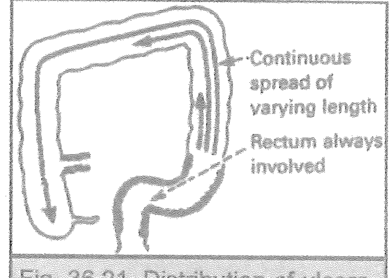
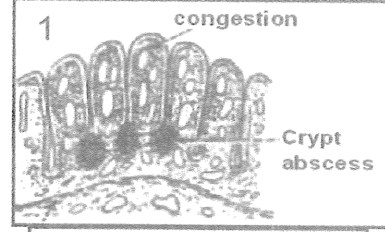


Fig. 36.21. Distribution of ulcerative colitis.

Pathology

- The disease begins at the dentate line of the anal canal and extends proximally. Extension is continuous with no skip areas of normal bowel (Fig. 36.21). The extent of the disease varies from rectal involvement alone (proctitis), to whole colon and rectum affection (pancolitis).
- Unlike Crohn's disease, only the mucosa and submucosa are involved. The mucosal affection varies from granularity to extensive ulceration. The characteristic histological feature is the formation of crypt abscesses in the depths of the glandular tubules with surrounding inflammation. The abscesses fuse to form ulcers (Fig. 36.22). Pseudopolyps are likely to be found.



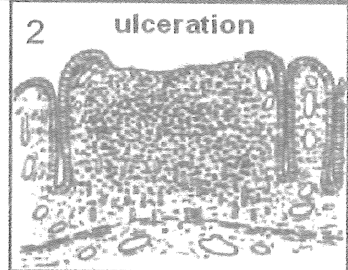


Fig. 36.22. Development of ulcerative colitis
1. Early phase with crypt abscess formation.
2. Established cases with ulceration.
The disease is MUCOSAL only.



Possible complications

- Intestinal
  - Toxic megacolon in 3-5% of cases. It can be fatal.
  - Haemorrhage.
  - Colon cancer. The risk is higher in patients with pancolitis for more than 10 years. Carcinoma on top of ulcerative colitis is often multicenteric.
- Extraintestinal complications include arthritis, uveitis, cholangitis, liver cirrhosis, and skin lesions as pyodemia gangrenosum, and erythema nodosum.

Clinical features

- The disease is more common in females.
- The commonest age incidence is the third and fourth decades.
- The patient commonly presents with watery diarrhoea mixed with blood, pus and mucus and accompanied by tenesmus.
- Pain, fever, weight loss, and dehydration occur to varying degrees.
- The disease is characterized by the occurrence of remissions, and relapses.
- Toxic megacolon. This is a very serious complication. There is severe, incessant diarrhoea with the passage of blood, mucous and pus in the stools. The patient is very toxic with severe pyrexia 39-39.5°C. Abdominal examination reveals severe abdominal distension due to marked colonic atony. Unless treated urgently, the



Fig. 36.23. Barium enema in a case of ulcerative colitis.

condition has a fatal outcome.

Investigations

1. Laboratory findings. Anaemia and leucocytosis in acute cases are present to varying degrees. Hypoproteinaemia and hypokalaemia may be present in severe cases.
2. Barium enema reveals mucosal irregularity and ulcers, as well as colonic shortening and loss of haustrations (Fig. 36.23) in long standing disease. It is contraindicated in the presence of toxic megacolon.
3. Colonoscopy and biopsy. It shows the mucosal abnormalities and detects the upper extent of the disease (Fig. 36.24). Endoscopy is contraindicated with toxic megacolon.

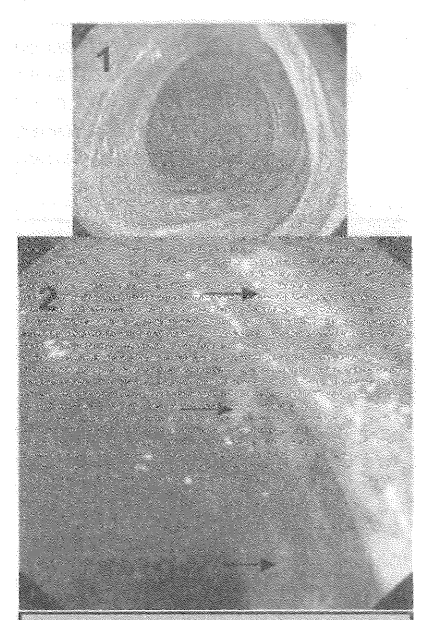


Fig. 36.24. Colonoscopy.
1. Normal colon
2. Ulcerative colitis. Multiple superficial ulcers (arrows) and congested mucosa between the ulcers.

Treatment

Medical treatment is indicated for the majority of cases.

- Correction of anaemia, hypoproteinaemia, and hypokalaemia by proper dieting, and supplementary vitamins and minerals.
- Aminosalysilates, e.g. sulphasalazine or mesalamine. The dose ranges from 2-6 gm/day according to the severity of the disease.
- Attacks of exacerbation are treated by corticosteroids, either systemic, or by enema in cases localized to the rectum. Oral administration of Flagyl is also recommended.

Surgical treatment

Indications

1. Failure of medical management to control the disease leading to a state of chronic ill health with persistent diarrhoea, anaemia and malnutrition.
2. Development of complications as toxic megacolon with perforation (urgent surgery), or colon cancer.
3. Long-standing pancolitis where biopsy shows dysplasia.
4. Stricture formation.

Surgical options

1. Excision of the whole colon and rectum (panproctocolectomy) with permanent ileostomy is the standard treatment. The patient may by reluctant to have this operation because he will be left with an ileostomy.
2. The colon is excised but the rectum may be spared and an ileo-rectal anastomosis is performed. Lifelong surveillance of the rectum is needed for fear of development of a rectal carcinoma.

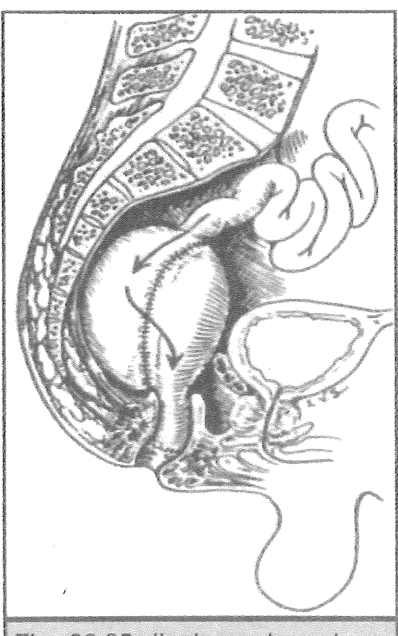


Fig. 36.25. Ileal pouch-anal anastomosis.

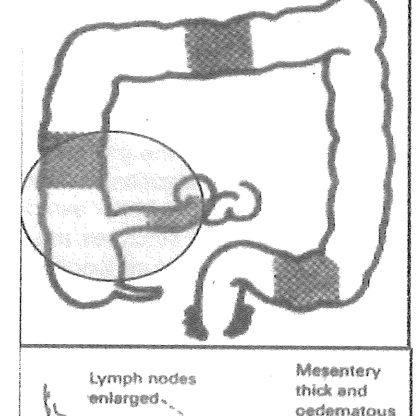
3. Proctocolectomy with distal rectal mucosectomy. Excision of the colon and the upper half of the rectum is performed. The mucosa of the lower part of the rectum, which is the site of the disease, is cored out leaving the muscle wall. An ileal pouch is fashioned to act as a reservoir, and is anastomosed to the anal canal within the preserved rectal muscles (Fig. 36.25). This sophisticated operation is not suitable in an emergency setting.

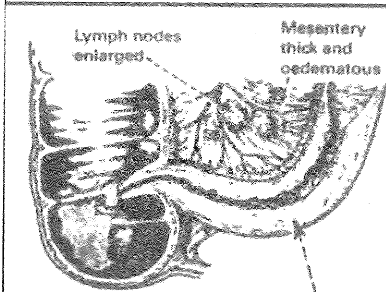
Crohn's disease

Crohn's disease (regional ileitis) is an idiopathic, chronic, transmural, granulomatous inflammatory bowel disease that can involve any area in the gastrointestinal tract.

Aetiology is unknown. Heredity may have a role. Disturbances in the immune system may be involved.

Pathology (Fig. 36.26)





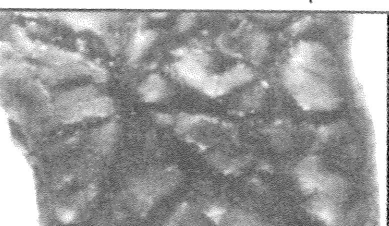
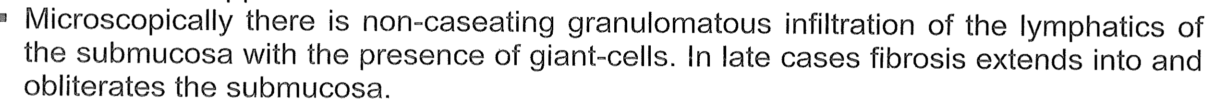


Fig. 36.26. Pathology of Crohn's disease.
1. Distribution.
2. Transmural affection.
3. Mucosal fissures that cause cobble stone appearance.

- The commonest site is the distal ileum; hence the old name regional ileitis. With the realization that the disease can affect any part from the mouth to the anal canal, this name was dropped from use. Next common site is the colon, followed by the anal canal.
- Crohn's disease is a chronic disease that is characterized by granuloma formation.
- The affected bowel segment is thickened as the inflammation involves the whole thickness from mucosa to serosa (transmural), as well as the mesentery which is also thickened and contains enlarged lymph nodes.
- In contrast to ulcerative colitis, Crohn's disease is characterized by skip lesions, i.e., totally healthy length of gut between the affected areas.
- In longstanding cases multiple fissures occur, between which the mucosa is oedematous giving a cobble stone appearance.



- Microscopically there is non-caseating granulomatous infiltration of the lymphatics of the submucosa with the presence of giant-cells. In late cases fibrosis extends into and obliterates the submucosa.

Complications

Intestinal

1. Stricture of the small intestine leading to intestinal obstruction.
2. Abscess and fistula to another bowel loop, bladder, vagina or to the skin.
3. Perianal abscess, anal fistula and fissure.
4. Malabsorption in extensive disease, or in those who had extensive intestinal resection.

Extraintestinal. Inflammatory ocular, joint, skin, and hepatobiliary lesions are similar to those of ulcerative colitis.

Clinical features

- The peak age of onset is in the second to fourth decades. Males and females are equally affected.

- Symptoms include diarrhoea, abdominal pain, weakness, weight loss, and anorectal disease. In old standing cases the patient may present with intestinal obstruction.
- Signs include anaemia, malnutrition, and possibly a mass in the right iliac fossa.

Investigations

1. Contrast radiography. Barium meal and follow through is done for suspected small intestinal disease while a barium enema examination is indicated for suspected colon disease. Radiography reveals segmental areas of stricture, separated by skip areas of free bowel, and cobble stone appearance of the mucosa caused by longitudinal fissures. It may also show internal or external fistulae. The finding of a narrowed terminal ileal segment is known as the string sign of Kantor" (Fig. 36.27).
2. Colonoscopy and biopsy of large bowel lesions proves the diagnosis.
3. Biopsy of suspected anal fissures and fistulae.

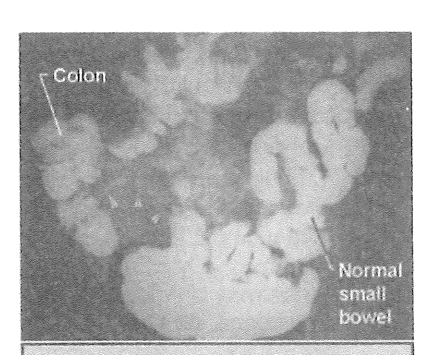


Fig. 36.27. Barium meal and follow through of Crohn's disease. It shows stricture of terminal ileum (white arrows) with proximal dilatation.

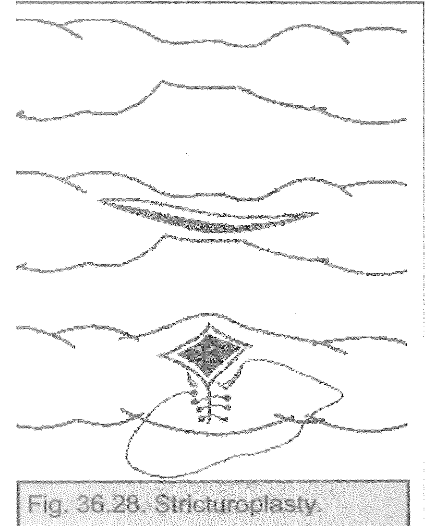
Treatment

Medical treatment

- Low residue, high protein and calorie diet, with supplementary vitamins and minerals.
- Antispasmodics are given for pain.
- Corticosteroids and antibiotics (sulphasalazine or flagyl) are also given in acute active disease but their value in preventing relapse is not established.
- Infliximab (antitumour necrosis factor antibody) helps closure of Cronh's disease fistulae.

Surgery is reserved for complications of the disease, but ultimately becomes necessary in most cases.

- The tendency is to perform limited resection to the area that is causing the problem. As the patient may be subjected to further resections in the future, removal of long intestinal segments is better avoided as it leads to malabsorption.
- Localized strictures are treated by stricuroplasty, i.e., incising the stricture longitudinally, and closing it transversely to widen the lumen (Fig. 36.28).


Fig. 36.28. Stricturoplasty.

Bilharzial colitis

The disease often affects young adult males in rural areas of Egypt.

Aetiology

The disease is caused by infestation with Schistosoma mansoni, and less commonly by Schistosoma haematobium.

Pathology

- The usual sites are the sigmoid colon and the rectum.
- Chronic inflammatory cells surround the deposited ova leading to the formation of Bilharzial granulomas.

- The disease may produce:
    - Polyps (Fig. 36.29)
    - Ulcers
    - Bilharzial pericolic mass
    -
- The condition is not precancerous and does not produce strictures. Possible complications, however, include:
    - Anaemia as a result of chronic blood loss.
    - Hypoproteinaemia as a result of chronic protein loss.
    - Partial rectal prolapse.
    - Prolapse and strangulation of a polyp.

Fig. 36.29. Bilharzial polyps of the colon.

Clinical features

- There is diarrhoea, tenesmus, and passage of mucus and blood in stools.
- Weakness, dyspepsia and colics are common.
- Pallor is commonly an evident feature.
- Clubbing of fingers is often noticed in association with ulcerated Bilharzial colon polyps.
- The sigmoid colon is palpably thickened and tender. In the diffuse form a hard nodular mass is felt in the left iliac fossa. The mass may be mobile or fixed, and is often so hard as to simulate cancer (but features of intestinal obstruction are absent).
- Rectal examination commonly detects multiple polyps.

Investigations

1. Laboratory
    a. Stool analysis shows blood, pus, and probably viable Bilharzia ova. Urine analysis may reveal associated urinary infestation.
    b. Blood picture may show anaemia.
    c. Liver function tests may reveal hypoalbuminaemia.
2. Sigmoidoscopy shows the mucosal changes. Examination of fresh biopsy specimens, or ulcer scrapings shows the ova.
3. Barium enema may exhibit multiple rounded filling defects of the polyps. The sigmoid colon may be pulled to the right iliac fossa.

Treatment

- All cases are initially treated by repeated courses of antibilharzial drugs. Oxamniquine or praziquantel are the most appropriate for treatment (Chapter 7).
- Polyps are treated by fiberoptic colonoscopic polypectomy at multiple sessions.

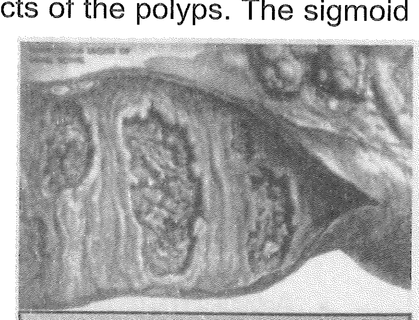


Fig. 36.30. Tuberculous ulcers have transverse orientation.

Intestinal tuberculosis

There are two forms of intestinal tuberculosis.

Ulcerative tuberculous enteritis is secondary to pulmonary tuberculosis, and arises as a result of swallowing tubercie bacilli. There are multiple ulcers in the terminal ileum. The long axis of a tuberculous ulcer lies transversely (Fig. 36.30). Perforation is rare, but stricture is a possibility.

Clinical features. Diarrhoea, abdominal pain, and weight loss are the predominant symptoms. In late cases the patient may develop intestinal obstruction due to stricture formation.

Treatment. In early cases treatment is by antituberculous drugs. In patients with intestinal stricture, surgical excision may be needed.

Hyperplastic ileocaecal tuberculosis is a primary infection due to ingestion of tubercle bacilli (bovine or human type) by patients with high resistance. The disease occurs most commonly in the ileocaecal region. There is much thickening of the intestinal wall, and narrowing of the lumen. There is early involvement of the regional lymph nodes, which may caseate.

Unlike Crohn's disease (which in many respects this disease simulates), abscess and fistula formation are rare. Untreated, intestinal obstruction supervenes.

Clinical features. Attacks of abdominal pain and intermittent diarrhoea are the main symptoms. Sometimes, the presenting picture is that of a mass in the right iliac fossa in a patient with vague ill health.

Investigations. A barium meal and follow through reveals narrowing of the lumen of the affected area, and an elevated caecum that may be subhepatic.

Treatment is by antituberculous chemotherapy. Right hemicolectomy with removal of the diseased iieum is indicated in obstructing lesions and if malignancy cannot be excluded.

Surgical complications of typhoid and paratyphoid

1. Intestinal haemorrhage as a result of ulceration of the affected Peyer's patches in the lower ileum. The ulcer is parallel to the long axis of the gut. Bleeding usually occurs 2-3 weeks after the onset of the disease. It usually presents as passage of dark blood per rectum, but may be bright red if bleeding is profuse.
2. Perforation of a typhoid ulcer may occur around the third week with resulting peritonitis. The condition calls for urgent exploration under cover of chloramphenicol. Pus is evacuated, and either the perforation is dosed or the diseased ileal segment is resected.
3. Acute non-calcular cholecystitis.
4. Chronic cholecystitis (carrier state).
5. Deep venous thrombosis of the lower limbs is a complication of prolonged recumbency.

Intestinal ischaemia

Chronic intestinal ischaemia

Chronic intestinal ischaemia is caused by narrowing of the superior mesenteric artery, and sometimes the coeliac artery. The disease affects elderly patients who are likely to have atherosclerosis of other vessels.

Aetiology

1. Atherosclerosis is the main cause. The intima is thickened at the mouth of the superior mesenteric artery causing narrowing.
2. Rarely the coeliac axis is compressed by the median arcuate ligament of the diaphragm.

Clinical features

- The main symptom is postprandial abdominal pain, which starts 15-30 minutes after a meal, and lasts for about an hour This pain is called "abdominal angina".
- The patient is usually afraid to eat, and, therefore, loses weight.
- An upper abdominal bruit is heared in the majority of patients.
- A clinical search is made to find out atherosclerotic narrowing of other arteries.

Investigations

Investigations are needed to:

1. Exclude other causes of chronic abdominal pain, e.g., ultrasound and upper GI endoscopy.
2. Confirm the narrowing of vessels. Aortography is diagnostic, particularly its lateral view which delineates the narrowing of the origin of the artery, as well as the presence of collateral circulation.
3. CT angiography is non-invasive and provides a 3D image.

Treatment

- Atherosclerosis is surgically treated either by endarterectomy (coring out the thickened intima), or by inserting a bypass dacron graft from the aorta to the superior mesenteric artery beyond the narrowing.
- Median arcuate ligament compression is treated by division of the ligament.

Acute intestinal ischaemia

Acute ischaemia of the intestine is a very serious surgical emergency that is highly fatal. A large segment of intestine is commonly affected, where there is evident occlusion of one or more of the mesenteric vessels. It is usually the superior mesenteric artery or vein that is occluded. On the other hand, ischaemia may be focal and affects a small segment of bowel.

Aetiology

Acute mesenteric vascular occlusion:

1. Mesenteric arterial embolism. The emboli usually arise from the heart. The source may be:
    a. Atrial fibrillation with a left atrial clot.
    b. Mural thrombus after myocardial infarction affecting the left ventricle. The embolus usually lodges within few centimeters of the mouth of the superior mesenteric artery, sparing the middle colic and a few of the upper jejunal branches.
2. Mesenteric arterial thrombosis. The condition complicates cases with mesenteric atherosclerosis. Patients usually give history of intestinal angina. The extent of ischaemia is more than that with embolism as the block is at the mouth of the superior mesenteric artery sparing none of its branches.
3. Mesenteric venous thrombosis. The condition is sometimes idiopathic or may be associated with the following disorders:
    a. Portal hypertension.
    b. Intra-abdominal sepsis.
    c. Hypercoagulable states.
    d. Intake of contraceptive pills.

Non-occlusive intestinal ischaemia

Non-occlusive intestinal ischaemia is associated with low cardiac output states as arrhythmias and major sepsis. Splanchnic vasoconstriction occurs; inducing intestinal

ischaemia that may lead to gangrene. There is no block to the mesenteric vessels, yet some of these patients have atherosclerotic narrowing.

Pathology

Damage to the intestine occurs from the ischaemia, and from the re-establishment of intestinal perfusion, the so called "reperfusion injury".

Ischaemic damage

1. The most sensitive layer to ischaemia is the mucosa. Within three hours of complete vascular block it sloughs, ulcerates and bleeds in the lumen. Bacteria can get access to the blood stream through the damaged mucosal barrier.
2. Within a few hours the whole thickness of the intestinal wall is affected, and exudes serosanguinous fluid in the peritoneum. The patient may lose a substantial amount of blood.
3. The intestine becomes gangrenous and soon perforates producing peritonitis.
4. The gangrenous bowel segment loses peristalsis and, thus, acts as an obstruction to the flow of intestinal contents. The proximal intestine becomes distended with fluid and gas. With the development of peritonitis, paralytic ileus sets in producing more generalized distension.

Reperfusion damage. The return of blood flow, either spontaneously or by surgery, results in the release of oxygen radicals that damage the cell membrane.

Clinical features

- Severe acute abdominal pain is the main symptom. Pain responds neither to narcotics nor to nasogastric aspiration.
- There is a variable degree of vomiting and of bleeding per rectum.
- In the early stage there are little physical signs to match the severity of pain. Later with the development of hypovolaemia from blood loss, and peritonitis from perforation, the patient may become shocked with abdominal tenderness, rigidity, and distension.

Differential diagnosis

- Acute pancreatitis. The common features are severe pain, vomiting, minimal local signs in the early phase, and shock. The distinction is important as the management of acute pancreatitis is essentially conservative, while that of acute mesenteric vascular occlusion is urgent surgery.
- Intestinal strangulation.

Investigations

Unfortunately there is no specific or sensitive test to prove the diagnosis. The most useful aid is to bear the condition in mind, particularly in the atherosclerotic or the patient with cardiac arrhythmia.

- Blood picture. There is marked leucocytosis. The haematocrit value is usually elevated, but it may be reduced in case of massive blood loss.
- Serum amylase is elevated in half the patients, but is not as high as in acute pancreatitis.
- Plain X-ray of the abdomen may show air-fluid levels in the proximal intestine. Intestinal necrosis may show lately as intramural gas or gas in the portal venous system.
- The value of arteriography is controversial, and its use may delay surgical intervention. However, if the arteriographic findings are diagnostic of superior mesenteric artery embolism, preoperative infusion of papaverine directly in the artery improves intestinal viability.

Treatment

Urgent surgery is the key to survival.

Preoperative measures

1. Restoration of blood volume by blood and crystalloid infusion.
2. Parenteral antibiotics.
3. Nasogastric decompression by a Ryle's tube.

Laparotomy is done to:

1. Resect gangrenous intestine. Primary anastomosis is better avoided in cases of peritonitis with friable tissues, doubtful viability of the remaining intestine, and in patients with bad general condition. In such cases the two bowel ends are brought out to the surface and the effluent is collected from the proximal end using an ileostomy bag. Bowel anastomosis is postponed until the general and local conditions improve. For patients who receive a primary anastomosis a second-look operation after 24 hours is advised by some surgeons to check the viability of the intestine.
2. Restore arterial blood flow in cases with reversible ischaemia. Revascularization by embolectomy for the embolism, or by endarterectomy or bypass for the acute thrombosis is usually done.
3. Mesenteric venous thrombosis is diagnosed at operation by the presence of oedema of the mesentery, and by the extrusion of clots when the mesenteric veins are cut. Gangrenous intestine is resected. The patient is given postoperative heparin, and is discharged home under oral anticoagulant therapy for at least three months to prevent recurrence.

Prognosis

- Mesenteric venous thrombosis has a mortality rate of 30%, while that of arterial occlusion is about 45%. Embolism has a slightly better prognosis than arterial thrombosis.
- Up to 70% of the small intestine can be resected without major nutritional consequences. Patients who have resection of longer segments develop the 'short bowel syndrome" whose main feature is malabsorption. Many months are needed for the intestine to accommodate by increasing its absorptive surface. Patients are initially fed on total parenteral nutrition for a few months, and later oral intake is gradually introduced, reducing the amount of parenteral feeding, until ultimately the patient can depend totally on normal oral feeding.

Ischaemic colitis

- Patients are usually 50-70 years old and have some chronic vascular disease of the mesenteric vessels. A drop of blood pressure for any reason is the usual precipitating factor, In addition, surgery on the abdominal aorta may lead to colonic ischaemic if the inferior mesenteric artery is sacrificed.
- The splenic flexure is the most commonly affected area. The patient complains of acute abdominal pain and the passage of blood per rectum.
- Plain X-ray may show 'thumb-prints' due to oedematous mucosa outlined by bowel gas.
- The consequences of ischaemia are:
    - Complete resolution.
    - Gangrene, perforation, and peritonitis.
    - Stricture.
- Treatment is supportive by intravenous fluids, analgesics, and antibiotics. Surgery is seldom required for gangrene or for stricture formation.

Intestinal Tumours

Small intestinal tumours

Small intestinal tumours are rare.

Benign tumours. These may cause intussusception or may bleed.

- Adenoma, submucous lipoma, and leiomyoma.
- Peutz-Jegher's syndrome consists of:
  - Multiple familial intestinal hamartomatous polyps mainly affecting the jejunum.
  - Melanosis of the oral mucous membrane and the lips.
  - The disease is not highly precancerous, therefore, only the complicated polyps (those causing bleeding or intussusception) require removal or excision of the affected intestinal segment.



Tubular adenoma of the colon

Malignant tumours. In order of frequency, these are:

- Non-Hodgkin's lymphoma.
- Adenocarcinoma.
- Carcinoid tumour of the small intestine which is less common than that of the appendix but tends to be more malignant.

Colorectal polyps

Epithelial
Neoplastic
- Adenoma, solitary or multiple (FPC and Gardner's syndrome)
- Carcinoma

Hamartomas
- Juvenile polyps, solitary or multiple
- Peutz Jegher's polyps

Inflammatory
- Ulcerative colitis (pseudopolyps)
- Bilharzial

Mesenchyma
Lipoma, leiomyoma, or haemangioma may project in the lumen as polyp

Colorectal tumours

Tumours of the colon and the rectum share common properties and are, therefore, commonly referred to as cobrectal tumours.

Benign tumours

Benign colorectal tumours usually form polyps.

Epithelial

Neoplastic

Solitary adenoma may be:

	Risk of malignancy
Tubular	5%
Tubulovillous	22%
Villous	40%

FPC and Gardner's syndrome

Hamartomas

Juvenile pobyps, solitary or multiple

Peutz Jegher's polyps

Mesenchyma

Lipoma, leiomyoma, or haemangioma may project in the lumen as polyp

Malignant tumours

Primary

- Carcinoma.
- Carcinoid tumour.
- Sarcomas.

Secondary. These are the result of invasion from a nearby malignant tumour.

Two of the above tumours deserve detailed consideration, these are familial polyposis coli (FPC), and carcinoma.

Familial polyposis coil (FPC)

Aetiology

FPC is an autosomal dominant disease. Approximately 50% of the children of affected parents have this disorder, and only individuals with polyps transmit the disease. Males are affected more than females.

Pathology

- The colon and rectum are full of multiple polyps (at least 100) which are sessile and pedunculated (Fig. 36.31). The sigmoid colon and rectum are the commonest sites of affection.
- Three histological types are recognized; the tubular, tubulovillous, and villous.
- Left untreated, carcinoma develops in 100% of the affected patients by the fifth decade. The malignant potential is related to the size, and to the type of the adenoma. The incidence of malignancy is highest with villous adenomas that are bigger than 2cm.
- Gardner's syndrome is a variant of FPC. In addition to the above, there are polyps in the rest of the gastrointestinal tract, osteomas of the mandible and skull, cysts, soft tissue tumours, and desmoid tumours of the abdominal wall.

Clinical features

- Polyps usually present between the ages of 10 and 15 years.
- The commonest symptoms are diarrhoea, bleeding, and abdominal pain.

Investigations

- Barium enema shows multiple rounded filling defects throughout the colon and the rectum (Fig. 36.32).
- Sigmoidectomy or colonoscopy (Fig. 36.33) and biopsy prove the nature of the disease.

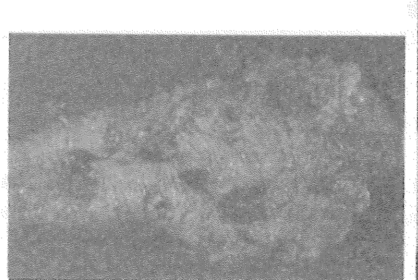


Fig. 36.31. Multiple polyps seen in an opened segment of colon.



Fig. 36.32. Barium enema showing multiple filling defects that are caused by multiple polyps.

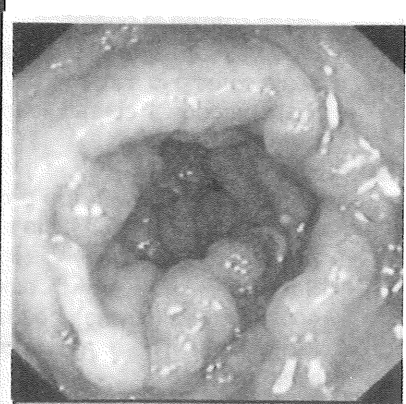


Fig. 36.33. Colonoscopic view showing multiple polyps.

Treatment

Treatment of the affected person is operative and is directed towards the eradication of the polyps. Surgical options are similar to those for ulcerative colitis and include:

- Excision of the whole colon and rectum (panproctocolectomy) with permanent ileostomy, is the standard treatment.
- The rectum may be spared and an ileoproctostomy is performed, with endoscopic fulguration of the remaining rectal polyps. Lifelong surveillance of the rectum is needed.
- Proctocolectomy with distal rectal mucosectomy, i.e., the mucosa of the lower part of the rectum is cored out leaving its muscle wall. An ileal pouch is fashioned to act as a reservoir, and is anastomosed to the anal canal within the remaining rectal muscles.

The family members should be periodically examined by colonoscopy and similarly treated if they develop polyps. The avoidance of an ileostomy encourages the asymptomatic person to have the operation. Genetic screening of the relatives is nowadays performed.

~10%
~65%

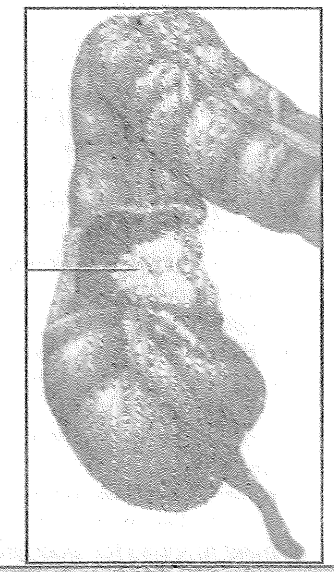
Fig. 36.34. Distribution of colorectal cancer.

Carcinoma of the colon and rectum

The disease is one of the leading causes of death from cancer in the Western society. The peak incidence is in the seventh decade of life.

Aetiology

Most colorectal cancers have an established genetic basis. The normal epithelium passes through several steps of genetic mutations to develop carcinoma (multi-step theory). Normal mucosa → dysplasia → adenoma → carcinoma.


Fig. 36.35. Cauliflower mass.

Risk factors and lesions

1. Solitary villous adenoma.
2. Familial polyposis coli, and Gardner's syndrome. As mentioned the risk is 100% in untreated cases. Cancer is more prone to develop in large adenomas that are larger than 2 cm particularly those of the villous variety.
3. Ulcerative colitis. The risk is highest with pancolitis of more than 10 years duration.
4. The disease is more common in Western countries probably due to lack of high fibre diet and increased animal fat.
5. Uretero-colic anastomosis.

Pathology

Microscopic picture

Histologically the tumour is an adenocarcinoma that arises from the columnar epithelium. It may be well, moderately, or poorly differentiated. Some tumours have a colloid structure, occur in younger patients, and have a poor prognosis.

Gross picture

Site (Fig. 36.34)

- Two thirds of cob-rectal cancer is situated in the rectum and the sigmoid colon.
- 10% occur in the caecum.
- The remainder are distributed over the rest of the colon.
- Multiple tumours occur in 5% of cases.

Gross types

1. Cauliflower-like polypoid variety arises more commonly in the caecum (Fig. 36.35).
2. Ulcerative type.
3. Stricture type arises more commonly in the sigmoid colon.

Spread

- Direct spread occurs first in the wall and then to the neighbouring organs, e.g., the urinary bladder. The strong fascia of Denonvillier lying in front of the rectum retards the spread of rectal cancer to the bladder.
- Lymphatic spread produces tumour deposits in the epicolic, paracolic, intermediate, and then superior or inferior mesenteric preaortic nodes. Nodal involvement is found in 40% of cases coming to operation.
- Blood stream spread produces liver metastases in 20% of patients coming to surgery.
- Transperitoneal spread leads to peritoneal nodules and ascites.

Staging

TNM staging is now commonly applied (Table 36.2)

Table (36.2): TNM staging of colorectal cancer

Tumour	T_1	Invades into submucosa
	T_2	Invades into musculosa
	T_3	Invades to the subserosa but not breaching the serosa
	T_4	invades serosa or another organ
Nodes	N_0	No nodes involved
	N_1	1-2 nodes involved
	N_2	3 or more nodes involved
Metastases	M_0	No metastases
	M_1	Metastases

Complications

- Intestinal obstruction occurs in 20% of cases particularly with left colon tumours. This tendency is attributed to:
  - The smaller lumen of the left colon.
  - Stool tends to be more solid.
  - Carcinoma tends to be of the stenosing variety.
- Perforation or the formation of an enterocolic or vesicocolic fistula.
- Bleeding. Chronic bleeding is the rule. Massive bleeding is rare.
- Complications due to spread, e.g., jaundice, liver failure, and ascites.

Clinical features

Cancer of the rectum is commoner in males, while cancer of the caecum is commoner in females. Clinical features depend upon the location of the tumour, its size, and the presence of metastases.

Right colon cancer (Fig. 36.37)

1. The usual presentation is vague with anaemia, weakness and loss of weight (Anaemia, Anorexia, Asthenia, 3A).
2. The patient may present with recurrent attacks of pain in the right iliac fossa.
3. A hard mass may be present in the right side of the abdomen. It is differentiated from appendicular mass by the long duration and absence of toxaemia and tenderness (differential diagnosis of a mass in the right iliac region).
4. The patient does not present by intestinal obstruction as the lesion is usually of the cauliflower variety, the contents are liquid and the lumen of the colon is wide. Obstruction occurs, rarely, if the lesion obstructs the ileocaecal valve.

Left colon cancer (Fig. 36.37)

1. The usual presentation is change of bowel habits, usually as progressive constipation, but there may be diarrhoea or attacks of constipation alternating with diarrhoea. These patients are usually diagnosed and treated as having colitis. Spurious diarrhoea (early morning slime) may be present.
2. Large bowel obstruction. The patient may present as acute, subacute or chronic large bowel obstruction. There is constipation, severe abdominal distension but vomiting is late. Carcinoma of the sigmoid colon is a very common cause of intestinal obstruction in an elderly patient.
3. Bleeding per rectum. Carcinoma of the left colon is a common cause of fresh bleeding per rectum but it is not a common cause of massive bleeding (compare with diverticular disease and angiomatous malformation).
4. Mass in the left side of abdomen. As the lesion is usually of the infiltrating schirrous variety, it rarely presents by a mass. If a mass is palpable, it is usually due to the impacted stools above the obstruction.

Rectal cancer

1. Rectal cancer often presents with bleeding per rectum which is usually slight. Whenever rectal bleeding occurs in a middle aged or older individual, even in the presence of haemorrhoids, coexisting rectal cancer must be ruled out.
2. There may be tenesmus (sense of painful incomplete evacuation), and the passage of mucus.

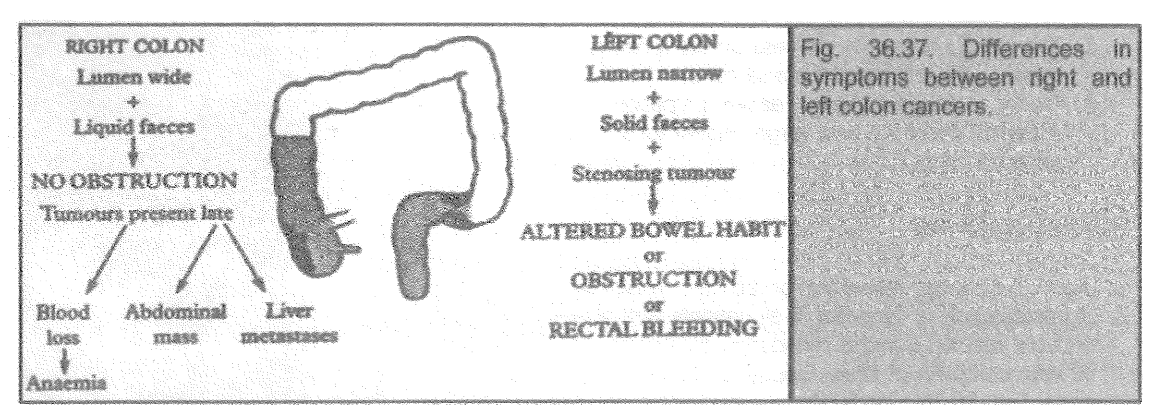


Fig. 36.37. Differences in symptoms between right and left colon cancers.

3. The tumour is usually painless unless it has spread outside the rectal wall or infiltrated the anal canal.

4. Digital rectal examination allows palpation of lesions that lie within 10 cm of the anal verge. Higher tumours are revealed by sigmoidoscopy.

Investigations

1. Blood picture may reveal microcytic hypochromic anaemia.
2. Sigmoidoscopy is essential in all patients with altered bowel habit or rectal bleeding, and is mandatory in patients above the age of .40 who complain of piles. Biopsy is obtained from suspicious lesions (Fig. 36.38). Biopsy from a higher tumour can be obtained by the colonoscope.
3. Barium enema. The tumour appears as a fixed irregular filling defect. Annular strictures of the left colon show a characteristic "apple core appearance" (Fig. 36.39). Even with a palpable rectal cancer, contrast radiography is useful to exclude a second higher tumour.
4. To detect spread liver function tests, abdominal ultrasound (or CT scan), and chest X-ray are done. If the tumour is expected to be close to the ureter, an intravenous urogram (IVU) is recommended.
5. MRI and endorectal ultrasound can assess the depth of invasion of rectal cancer.
6. Carcinoembryonic antigen (CEA) is a tumour marker whose serum level is high in colorectal cancer but is not specific. It is of prognostic rather than diagnostic value. The level drops after a successful radical surgery. If it shows a rise in the follow up period, this signifies recurrence.
7. For patients presenting with acute intestinal obstruction plain X-ray of the abdomen, blood picture, blood urea, and electrolytes are needed. A barium enema in these circumstances can assure the diagnosis.



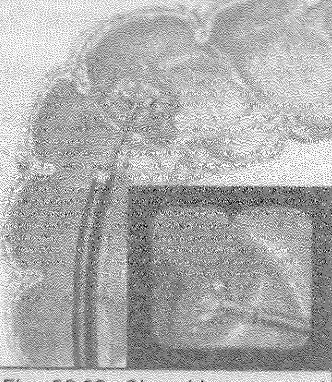


Fig. 36.38. Sigmoidoscopy and biopsy of sigmoid cancer.

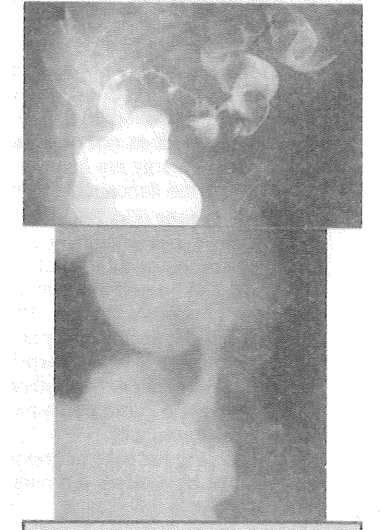


Fig. 36.39. Barium enema. Upper radiogram shows an irregular filling defect of a recto-sigmoid cancer. The lower one shows apple core appearance.

Treatment

General principles

- Surgery is the main line of treatment. Radical resection is the only curative measure. For inoperable tumours resection also offers the best palliation.
- Treatment depends on whether the tumour presents by acute obstruction or not and whether the tumour is operable or not.
- Inoperability can be judged before the operation, e.g., by the presence of liver metastases on ultrasound examination. Still tumours that are preoperatively judged to be operable may prove inoperable during exploration, because of liver metastasea, peritoneal nodules, or fixation of the tumour to important irremovable structures (irresectable tumour).

No acute obstruction

Operable (potentially curable) cases should receive elective radical resection. The cperation aims at cure and entails removal of the tumour bearing segment together

with its lymphatic drainage area, in one mass; and then to restore bowel continuity. Since the lymph nodes are so close to the main blood vessels arising from the superior and inferior mesenteric, their clearance requires ligation and division of these vessels at their origin, and consequently the whole part of the colon supplied by the removed vessels should be resected.

- Preoperative colon preparation allows a safer anastomosis.
- Tumours of the caecum require a right hemicolectomy where the ileocolic, and right colic vessels are divided at their origin. The terminal 10 inches of the ileum, the caecum, the ascending colon, the hepatic flexure, and the right half of the transverse colon are resected. The peritoneum of the posterior abdominal wall between the colon and the superior mesenteric that contains the divided arteries and veins, and the related lymph vessels and nodes, is removed in continuity. The operation is completed by performing an ileotransverse anastomosis (Fig. 36.40).

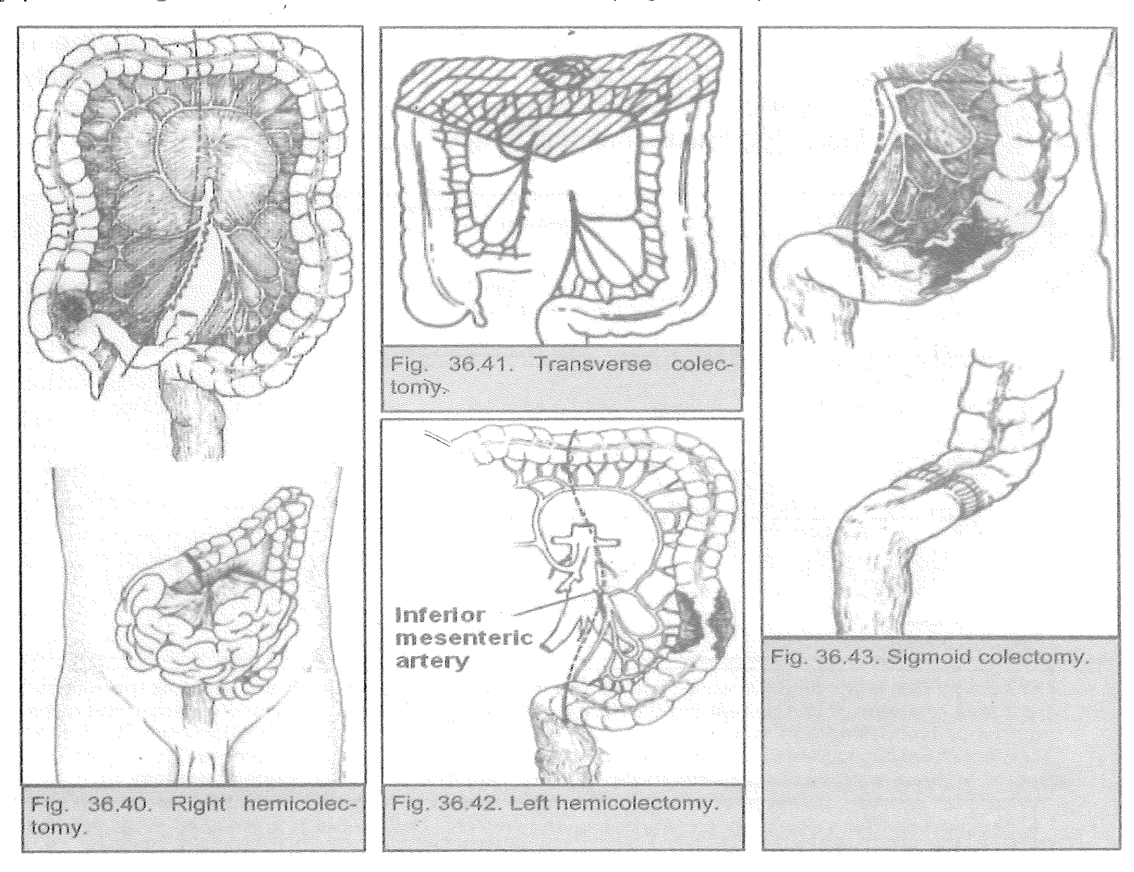


Fig. 36.40. Right hemicolectomy.

Fig. 36.41. Transverse colectomy.

Fig. 36.42. Left hemicolectomy.

Fig. 36.43. Sigmoid colectomy.

- Tumours of the ascending colon or hepatic flexure are treated by extended right hemicolectomy, where in addition to the above, the middle colic vessels are included. The resection extends to the junction of the right two thirds and the left third of the transverse colon.
- Tumours of the transverse colon are treated by transverse colectomy where the middle colic vessels are divided flush at their origin and the transverse colon is resected with both the hepatic and splenic fiexures, the transverse mesocolon, and the omentum (Fig. 36.41).
- Tumours of the left colon are treated by left hemicolectomy, where the inferior mesenteric vessels are divided at their origin and the resection extends down to the rectum (Fig. 36.42).

- Tumours arising in a long redundant sigmoid colon may be treated by sigmoid colectomy, where the sigmoid vessels are divided at their origin from the inferior mesenteric (Fig. 36.43).
- Tumours of the upper third of the rectum are treated by an anterior restorative resection where the inferior mesenteric artery is divided (or sometimes the left colic is spared) and the sigmoid colon, and the upper half of the rectum are excised. Bowel continuity is then restored (Fig. 36.44).
- Tumours of the lower third of the rectum are treated by an abdominoperineal resection (of Miles). It is similar to the above but the whole rectum is excised together with the anal canal, and a terminal colostomy is fashioned. The operation is called abdominoperineal because part of it is done through the abdomen and the other through the perineum (Fig, 36.45).
- Tumours of the middle third of the rectum used to be treated by abdominoperineal resection and a colostomy like lower third tumours. Two factors made a change in this policy towards anterior restorative resection. It is now realized that in the rectum inclusion of a 2 cm safety margin below the tumour is compatible with radicality, thus leaving enough distal rectum for anastomosis. The development of the circular staplers markedly facilitated this technically difficult procedure (Fig. 36.44).

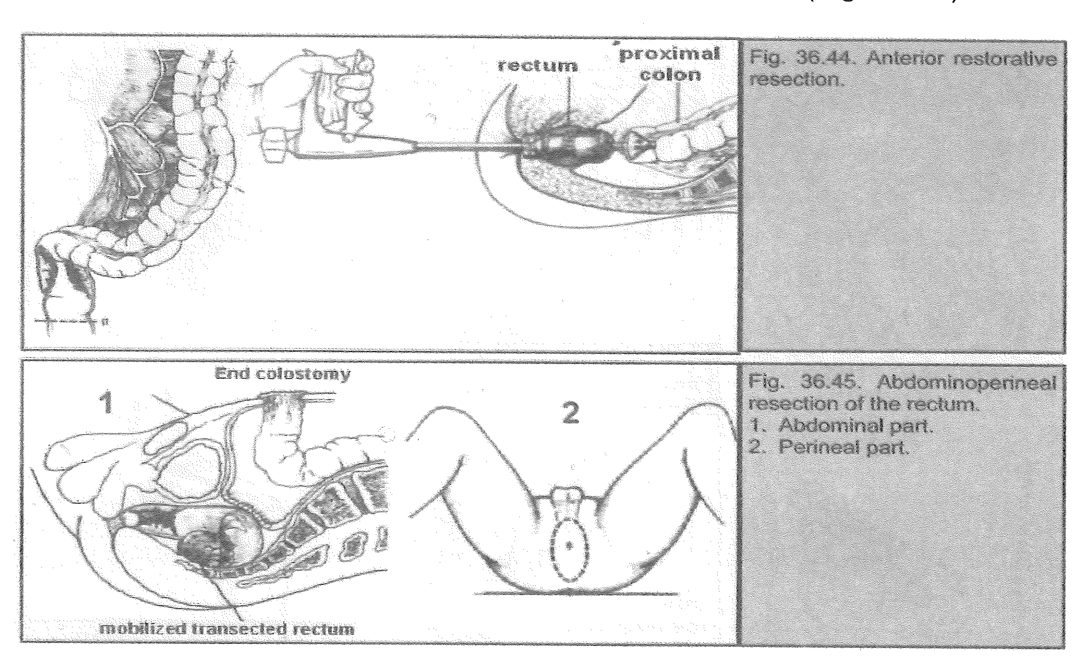


Fig. 36.44. Anterior restorative resection.

Fig. 36.45. Abdominoperineal resection of the rectum.
1. Abdominal part.
2. Perineal part.

Inoperable (incurable) cases (whether inoperability is diagnosed before or during operation).

- Whenever possible, palliative resection of the colon cancer is preferred. Here there is no need for wide resection of the bowel nor its lymphatics and vessels. The operation is destined to obviate the risk of obstruction and bleeding.
- If the tumour is irresectable, an operation is done just to avoid obstruction.
    - Tumours of the right colon require a side to side ileotransverse anastomosis.
    - Tumours elsewhere in the colon require a proximal colostomy.
    - Tumours of the rectum may show partial response to local radiotherapy.
    - Supplementary chemotherapy with 5-fluorouracil (5-FU) may be useful for colon cancer.

Acute intestinal obstruction

Urgent surgery is required after adequate preparation (see under intestinal obstruction).

- The best is colon resection (radical or palliative depending on operability), and colostomy for later anastomosis (see under principles of colon surgery). If it were a lesion in the right colon, which is rare to obstruct, primary anastomosis is feasible.
- For the critically ill, a proximal colostomy is all that can be done.

Worse prognosis of colorectal cancer is encountered in the following situations:

- Young age at clinical presentation.
- Colonic obstruction or perforation.
- Blood vessel or lymphatic invasion.
- Liver and/or distant metastases.
- Aneuploidy.

Intestinal obstruction — Classification

Intestinal obstruction is defined as arrest of downward propulsion of intestinal contents. There are different types of classification of intestinal obstruction.

According to the pathological nature of cause:

1. **Simple mechanical** obstruction is caused by an organic block. It produces acute abdominal pain.
2. **Strangulation** obstruction is characterized by significant impairment of the blood supply of the involved bowel segment. The ischaemia may result from twisting of the intestinal blood supply upon itself (volvulus), or from constriction of the blood flow by a tight band or a hernia opening. It may also result from thrombosis or embolism of the mesenteric vessels. If strangulation is not relieved within 6 hours, it will lead to gangrene.
3. **Paralytic ileus** is due to loss of propulsive power of the bowel leading to functional obstruction.

According to the level of obstruction:

1. High small bowel obstruction.
2. Low small bowel obstruction.
3. Large bowel obstruction.

According to the onset and course of obstruction:

1. **Acute obstruction**. The clinical course is rapid and the symptoms are early to develop.
2. **Chronic obstruction**, e.g., colon cancer, the symptoms are insidious and slowly progressive. The patient has constipation and distension.

A chronic obstruction may develop acute symptoms as the obstruction suddenly becomes complete when a narrowed lumen becomes totally occluded by dry stools, a condition that is termed acute on top of chronic obstruction.

Acute mechanical intestinal obstruction — General principles

Aetiology

Causes

1. In the lumen, e.g., faecal impaction, gallstone and parasitic infestation.

2. In the wall, e.g., congenital atresia, tumours, Crohn's disease, chronic diverticulitis and mesenteric vascular occlusion.
3. Outside the wall, e.g., adhesions (commonly post-operative), strangulated hernia, and volvulus.

- The most common cause of small bowel obstruction in adults is adhesions.
- The most common cause of small bowel obstruction in children is strangulated hernia.
- The most common cause of large bowel obstruction is colon cancer.

Common causes according to age

- **Neonates**. Congenital atresia, volvulus neonatorum, anorectal malformations, mechonium ileus, and Hirschsprung's disease.
- **Infants**. Ileocaecal intussusception, Hirschsprung's disease, and strangulated hernia.
- **Adults**. Adhesions, and strangulated hernia.
- **Elderly**. Colon carcinoma, and strangulated hernia.
- Strangulated hernia is a common cause in different age groups.

Pathology

Simple obstruction (Fig. 36.46)

- Distal to the obstruction the intestine empties and becomes collapsed.
- Proximally the intestine becomes distended by gas and fluid. Gaseous distension is due to swallowed air (68%), diffusion from congested vessels (22%) and bacterial fermentation (10%). Gastrointestinal secretions which amount to 8 litres per day accumulate above the site of obstruction. As the intraluminal pressure rises, absorption ceases.
- The stretched smooth muscles undergo hyperperistalsis (increased power and frequency of contractions) in an attempt to overcome the obstruction.
- Distension impairs blood supply, and may end in ulceration and perforation. This is evident in cases of colon obstruction with a competent ileocaecal valve, where the rising pressure in the closed proximal colon segment (closed loop obstruction) causes perforation of the caecum (Fig. 36.47). Perforation may also occur from the pressure of an adhesion band or the edge of a hernia defect on the bowel wall, producing local ischaemic necrosis.

Fig. 36.46. Simple intestinal obstruction.

Strangulation obstruction. In addition to the above:

- Bacteria and toxins in the lumen can transgress ischaemic bowel to the peritoneal cavity. Unrelieved strangulation can lead to septicaemic shock.
- The mucosa is the first layer to suffer from ischaemia producing acute ulceration and intra-luminal bleeding. If a long bowel segment is involved, blood loss may be substantial.
- Unrelieved strangulation is followed by gangrene of the ischaemic bowel with perforation and peritonitis.

General lethal effects

- Fluid and electrolyte loss from vomiting and from accumulation in the proximal bowel.
- Septicaemia from peritonitis.

Clinical features

Symptoms. The following are the cardinal symptoms of acute mechanical obstruction, but are not necessarily present in all cases.

1. Pain is usually the first symptom. It is colicky and is caused by the hyperperistalsis.
2. Distension is marked in colon obstruction and is present mainly in the flanks. It is less marked or absent in high obstruction. In low small bowel obstruction central abdominal distension occurs.
3. Absolute constipation is failure to pass flatus in addition to stools. It is an early symptom in colon obstruction but is late in high obstruction.
4. Vomiting occurs early in high obstruction but it is late or even absent when the colon is obstructed. In neglected cases vomiting becomes greenish then brown and offensive, i.e., faeculent (not faecal).

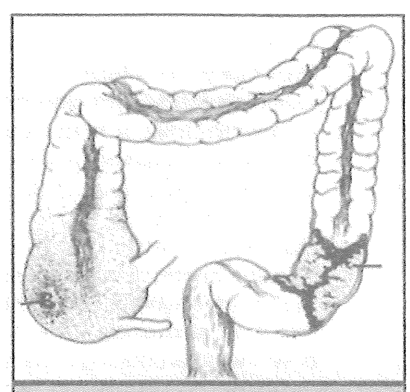


Fig. 36.47. An obstructing colon cancer may cause perforation of the caecum.

Examination

- General examination. Evidence of dehydration as tachycardia, oliguria, dry tongue, or even hypotension may be present.
- Abdominal inspection
    - Distension and visible peristalsis.
    - Strangulated external hernia should be looked for.
    - Scars of previous abdominal surgery may denote intraabdominal adhesions.
- Abdominal palpation. A mass may be felt (tumour or intussusception).
- Auscultation. Accentuated intestinal sounds.
- Rectal examination reveals an empty rectum. An exception is the finding of a hard faecal mass in an elderly bed-ridden patient causing faecal impaction.

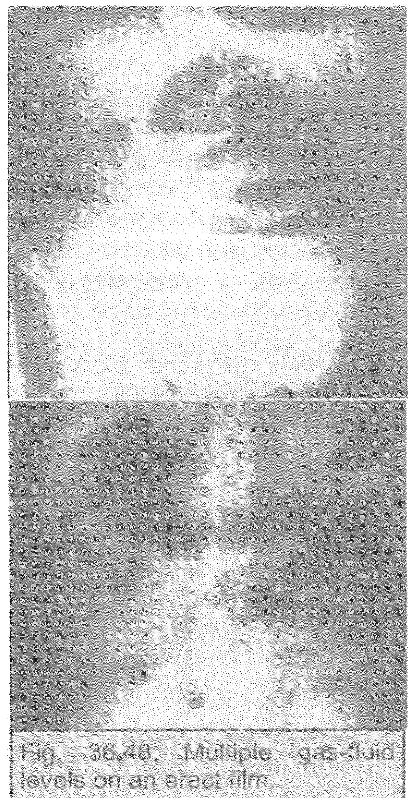


Fig. 36.48. Multiple gas-fluid levels on an erect film.

Strangulation is suspected with certain findings that indicate the necessity of urgent surgery.

1. Toxic patient, tachycardia, fever, and leucocytosis.
2. Evidence of blood loss as pallor, tachycardia, and hypotension.
3. Pain that is not relieved by naso-gastric suction. Pain is partly caused by hyperperistalsis, and partly by the ischaemia. Decompressing the bowel does not relieve ischaemic pain.
4. Marked tenderness, rebound tenderness, and rigidity.

Clinical assessment answers the following questions

1. Is there intestinal obstruction? Intestinal obstruction is diagnosed by the presence of colicky abdominal pains, distension, Vomiting and absolute constipation.
2. What is the pathological type of the obstruction? Strangulation is suspected when pain is severe, when there is toxaemia, when there is abdominal tenderness and

rigidity and by failure of bowel decompression to improve the pain. In paralytic ileus there is no pain and the bowel sounds are absent.

3. **What is the level of obstruction?**
    a. In high small bowel obstruction vomiting and dehydration are early. There is slight abdominal distension.
    b. In low small bowel obstruction, vomiting is delayed for about 12 hours, and there is central abdominal distension.
    c. In colonic obstruction, constipation is early, while vomiting may be absent or occurs after few days. Distension is marked especially in the flanks.
4. What is the cause of obstruction? The cause of obstruction is suspected from the age of the patient and by the presence of certain physical signs, e.g., presence of an appendicectomy scar points to adhesive intestinal obstruction.

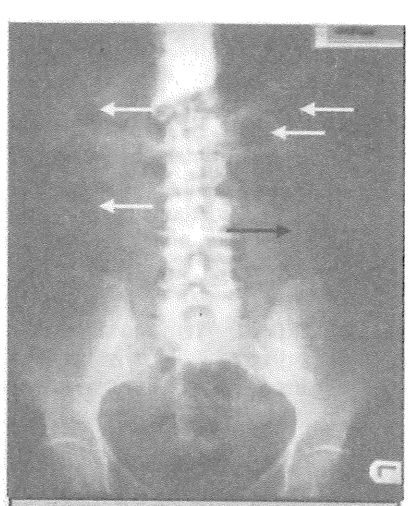


Fig. 36.49. Supine film in a case of distal small bowel obstruction. Distended jejunum shows valvulae conniventes (black arrow), while distended ileum shows as distended tubes with no mucosal pattern (white arrows).

Investigations

Investigations are needed to confirm the diagnosis of intestinal obstruction, to define its level and cause, and to estimate the severity of water and electrolyte imbalance.

1. **Plain X-ray of the abdomen**. Multiple gas-fluid levels on an erect film confirm the case as intestinal obstruction (Fig. 36.48). On a supine film the level of obstruction is known by the gas pattern of the distended proximal intestine, Distended jejunal loops show the characteristic circular mucosal folds (valvulae conniventes) crossing from one side of the lumen to the other. Distended ileal loops appear as featureless tubes with no mucosal pattern (Fig. 36.49). A colon full of gas shows haustrations that do not appear to reach the other side of the lumen.
2. Blood urea and electrolytes.
3. Blood picture.
4. Ultrasound examination can reveal distended bowel loops. A mass of intussusception can also be demonstrated.
5. CT scan with contrast has a sensitivity of 80-90% for the diagnosis of bowel obstruction.

Treatment

The treatment is urgent relief of obstruction, usually by surgery, after adequate preoperative preparation.

Pre-operative preparation "Drip and Suck"

- Intravenous replacement of fluid and electrolytes together with blood and plasma if the patient is shocked. Ringer's lactate solution is needed.
- Gastric aspiration by a nasogastric tube to decompress the bowel and to reduce the risk of inhalation during induction of anaesthesia.
- Antibiotics are given if there is a possibility of strangulation.
- A Foley catheter is inserted to check the urine output.

Operation

- A longitudinal exploratory incision is performed. An exception is the case of strangulated hernia where the incision is placed directly over it.
- The first step in exploration is to look at the caecum. If it is collapsed, this denotes small gut obstruction. If it is distended, this means large bowel obstruction.
- Determine the level of obstruction which is the junction of dilated and collapsed bowel loops.
- Relief of obstruction is attained by division of adhesive bands, reduction and repair of a hernia, or untwisting of volvulus.
- Bowel viability is assessed. Non-viability is known by:
    - Loss of peristalsis,
    - Loss of normal lustre.
    - Colour change; greenish or black bowel is non-viable, while purple bowel may still recover.
    - Loss of pulsations in the mesentery.
- Doubtful bowel may recover after relieving the obstruction if wrapped in .hot packs for a few minutes:
    - Non-viable intestine is resected.
    - Small intestinal ends are anastomosed (primary anastomosis).
    - Colon (see principles of colon surgery).

Conservative treatment may be successful in certains situations provided that the case is early and there is no clinical evidence of intestinal ischaemia.

1. Adhesive intestinal obstruction may be relieved by IV drip and nasogastric suction. Failure of conservative treatment and the suspicion of strangulation are indications for surgery.
2. Ileocaecal intussusception may be reduced by the hydrostatic effect of a barium enema and the reduction is radiologically monitored on the screen.
3. Sigmoid volvulus. Untwisting may be attempted using a rectal tube passed through a sigmoidoscope.
4. Faecal impaction is treated by enema to dissolve the obstructing hard faecal mass.

Acute mechanical intestinal obstruction - Special forms

Neonatal intestinal obstruction

Causes of intestinal obstruction in the neonatal period will be discussed in Chapter 37.

Intussusception

Definition

Intussusception is invagination of an intestinal segment (intussusceptum) into the lumen of an adjacent one (intussuscepiens). An intussusception is composed of (Fig. 36.50):

- An inner tube and a returning tube   Intussusceptum
- An outer tube   Intussuscepiens

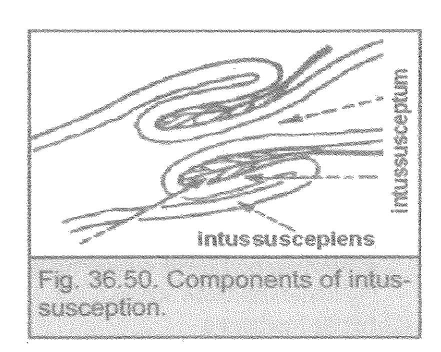


Fig. 36.50. Components of intussusception.

The blood supply of the inner layers of the intussusception is liable to be impaired at the neck of the intussusception.

Types

1. Ileo-ileal. A loop of ileum invaginates itself into an adjacent ileal loop.
2. Ileo-caecal. The termiflal ileum invaginates into the colon with the ileo-caecal valve repressing the apex of the intussusception (Fig. 36.51). This is the commonest type and usually affects infants.
3. Ileocolic. An ileal loop invaginates into an ileal loop and then passes to the colon through the ileocaecal valve. The blood supply of the ileal loop is tightly compressed by the ileo-caecal valve.
4. Colo-colic, a loop of colon invaginates into an adjacent colonic segment.

Aetiology

- The infantile ileocaecal intussusception is idiopathic. It is possible that an adenovirus causes swelling of the lymphoid follicles in the terminal ileum, and these protrude into the lumen acting as a foreign body which is forced distally along the gut. This may explain the frequent occurrence of intussusception at the age of weaning. The peak incidence is in summer due to increased gastroenteritis.
- In a minority of cases there is an evident cause at the head of the intussusceptum, e.g., polyp, Meckers diverticulum, or submucous haematoma in a patient with Henoch-Schonlein purpura. This occurs mainly in adults.

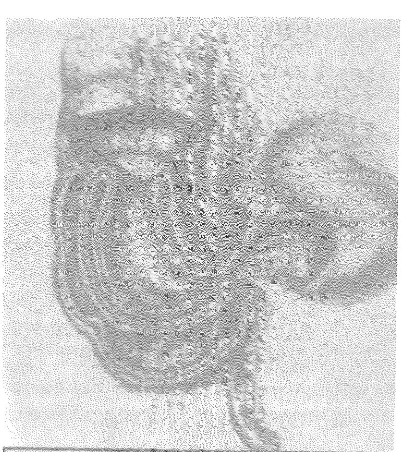


Fig. 36.51. Ileo-caecal intussusception.

Clinical picture of infantile ileocaecal intussusception

Symptoms

- The condition usually affects well nourished infants at the age of 3-12 months (age of weaning). The male to female incidence is 2:1.
- The infant awakens from sleep by severe abdominal colics, screams and draws his knees up onto the abdomen. These attacks alternate with intervals of apparent well-being during which the infant asks for feeding.
- With each attack of pain there is pallor, apathy and lethargy.
- Vomiting follows the attacks of colics in 85% of cases.
- The infant passes mucous and blood per rectum (red currant jelly stools).

Signs

- Emptiness in the right iliac fossa (Signe de Dance).
- Distension is usually absent in early cases. If it occurs it denotes possible perforation or gangrene.
- A sausage shaped mass may be felt.
- Digital rectal examination reveals bloody mucous in 60% of cases. Sometimes the head of the intussusception can be felt.

Investigations of infantile ileocaecal intussusception

1. Blood picture shows anaemia.
2. Ultrasound examination.
3. Barium enema: In cases of doubtful diagnosis a barium enema is performed. The barium will fill the colon until it stops at the area of intussusception where there is arrest of further progress of the contrast and a cylindrical filling defect appears with a trace of barium on either side (claw sign, Fig. 36.52).

Treatment of infantile ileocaecal intussusception

- Resuscitation of the infant by I.V. infusion of dextrose and saline. Antibiotics are prescribed. A nasogastric tube is inserted.
- In early cases a trial of hydrostatic reduction is performed. The pressure should not exceed 120cm of water.
    - Success of reduction is confirmed by free flow of barium into the small intestine for more than 5cm and by prompt clinical improvement with no further colics,
    - The baby should be kept under observation for 24 hours.
    - Success rate of hydrostatic reduction is 75-95%.
    - Contraindications to hydrostatic reduction:
        - Doubtful diagnosis.
        - Late cases.
        - Presence of abdominal distension or rigidity.
- Surgery is needed when hydrostatic reduction fails or the condition is advanced from the start. At laparotomy, the head of the intussusception is squeezed backwards out of the containing colon. The proximal ileum should never be pulled backwards to disengage the intussusception as this may lead to intestinal tears. The presence of gangrene or an irreducible intussusception is an indication for bowel resection and anastomosis.

> Any infant having colicky abdominal pains with the passage of blood-stained mucous per rectum should be suspected of having intussusception.



Fig. 36.52. Barium enema showing the characteristic claw sign in a case of infantile ileo-caecal intussusception.

Prognosis of infantile ileocaecal intussusception

Mortality is high in gangrenous cases.

Intussusception may recur in 2% of cases.

Adhesive intestinal obstruction

Intraperitoneal adhesions (Fig. 36.53) constitute the commonest cause of intestinal obstruction in adults in developed countries.

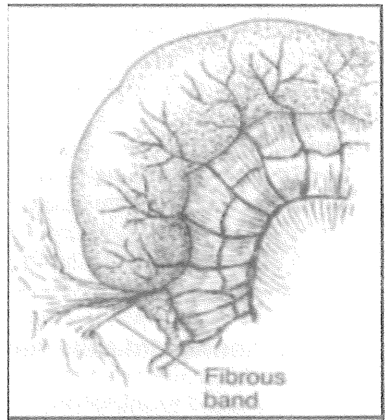


Fig. 36.53. Adhesive intestinal obstruction.

Aetiology

- Postoperative adhesions are the commonest. They result from previous abdominal surgery. Their exact aetiology is obscure. It is suggested that adhesions develop at the sites of ischaemia of the bowel or at the scars of the abdominal wall.
- Post-inflammatory adhesions may follow previous septic or tuberculous peritonitis.

Pathology

- Adhesions may be multiple or solitary.
- They bind one intestinal loop to another, or to abdominal wall.
- They induce obstruction by kinking or by directly obstructing a small intestinal loop.
- Strangulation may result from compression of the loop's blood supply. A band may induce localized ischaeniic necrosis by direct pressure on the intestine at the level of block.
- Adhesions have a tendency for recurrence producing recurrent intestinal obstruction.

Clinical features

- Acute or recurrent acute small bowel obstruction (colicky abdominal pain, vomiting, distension and absolute constipation).
- Almost always there is a scar of previous abdominal surgery.
- There are may be physical findings of strangulation.

Treatment

Conservative treatment may be tried in early cases with no evidence of strangulation, particularly if it is a recurrent obstruction.

- Naso-gastric suction and intravenous replacement of losses (drip and suck) constitute the main lines of conservative treatment.
- Close observation is necessary to judge success which is indicated by resolution of pain and distension, passage of flatus, and the retrieval of clear gastric aspirate.
- Conservative treatment should not be prolonged if there is no response in 48 hours.

Surgery is needed if the above trial fails, or if there is evidence of strangulation or gangrene. Adhesions are divided and the bowel viability is assessed and dealt with accordingly.

Strangulated hernia (Chapter 41).

Volvulus

Pathology (Fig. 36.54)

- Volvulus is twisting of a bowel loop around its mesenteric axis.
- Volvulus produces a combination of obstruction, of the closed- loop type and occlusion of the main vessels at the base of the involved mesentery (strangulation).
- What adds to the seriousness of the condition is that in a closed loop the pressure rises rapidly, further increasing the risk of gangrene and perforation.
- Volvulus is commonest in the sigmoid colon. It may also affect the caecum, stomach, or the small intestine. Midgut volvulus of the neonates is described in chapter (37).

Predisposing factors to sigmoid volvulus. Sigmoid volvulus is commoner in elderly chronically constipated males.

1. A long sigmoid colon.
2. A narrow base of sigmoid mesocolon.
3. A heavily loaded sigmoid as a result of chronic constipation.
4. Adhesions at the apex of the sigmoid loop facilitate its twisting.

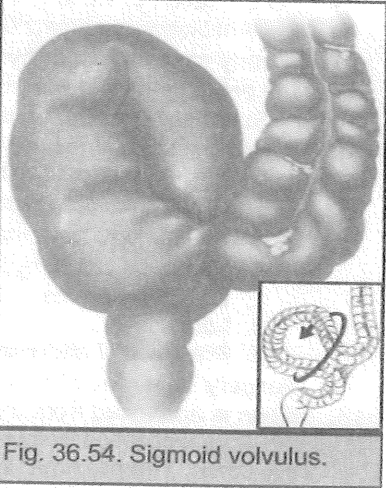


Fig. 36.54. Sigmoid volvulus.

Clinical features of sigmoid volvulus

- The features are those of acute colon obstruction with marked distension because of the accumulation of fluid and gas in the closed sigmoid loop.
- Neglected cases show evidence of peritonitis.

Investigations

1. Plain X-ray shows the huge gas-filled sigmoid loop that may look like the inner tube of a car tyre (omega loop, Fig. 36.55). The base of the distended loop points to the left lower abdomen.
2. Urea, electrolytes and blood picture.

Treatment

- **Conservative treatment** may be attempted in early cases with no evidence of gangrene. A rectal tube is passed through a signioidoscope to untwist the sigmoid loop. Success is rewarded by a gush of gas and fluid stools. The tube is left in place and the patient is prepared for later elective resection of the long sigmoid to prevent recurrence.
- **Surgery** is usually resorted to because of failure of the above or because of late presentation. Gangrenous bowel is resected, the proximal colon end is brought out to the skin as a terminal colostomy and the distal end is closed by sutures (Hartmann's procedure); for later elective anastomosis. Viable sigmoid is untwisted and either fixed to the posterior abdominal wall or resected as for gangrenous cases.



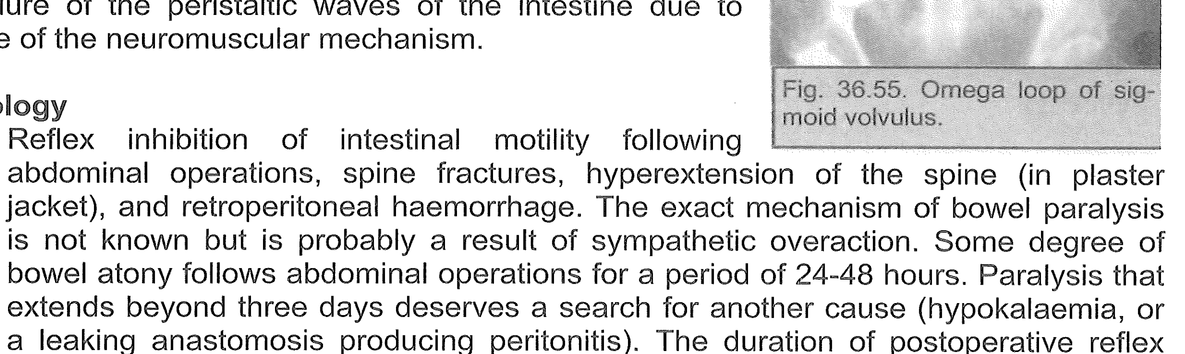


Fig. 36.55. Omega loop of sigmoid volvulus.

Paralytic ileus

This is a form of adynamic obstruction in which there is failure of the peristaltic waves of the intestine due to failure of the neuromuscular mechanism.

Aetiology

1. Reflex inhibition of intestinal motility following abdominal operations, spine fractures, hyperextension of the spine (in plaster jacket), and retroperitoneal haemorrhage. The exact mechanism of bowel paralysis is not known but is probably a result of sympathetic overaction. Some degree of bowel atony follows abdominal operations for a period of 24-48 hours. Paralysis that extends beyond three days deserves a search for another cause (hypokalaemia, or a leaking anastomosis producing peritonitis). The duration of postoperative reflex ileus depends on bowel manipulation and irritation by blood, bile, urine, or pus.
2. Metabolic abnormalities as hypokalaemia, uraemia, and diabetic ketoacidosis.
3. Peritonitis induces paralytic ileus due the direct toxic effect on the nerve plexuses of the intestine. The picture may be further complicated by the formation of fibrinous adhesions that kink the intestinal loops, thus adding an element of mechanical obstruction.
4. Drugs as anticholenergics (e.g., probanthine), and tricyclic antidepressants may produce paralytic ileus if taken in large doses.

Pathology

- There is marked distension of the small and large intestines with gas and fluid.
- The patient suffers a severe loss of fluid and electrolytes in the distended bowel and through vomiting.

Clinical features

- Paralytic ileus is most common after major abdominal surgery.
- Symptoms. Abdominal distension, absolute constipation, and effortless vomiting are the main symptoms. The patient has no colicky abdominal pains but there is only a sense of fullness and discomfort.
- Signs. Abdominal distension and inaudible intestinal sounds (silent abdomen) are the main signs. There may be evidence of localized or generalized peritonitis.

Investigations

1. Plain X-ray of the abdomen shows multiple gas fluid levels. The gaseous distension includes the whole small and large intestines.
2. Blood urea, electrolytes, and blood picture.

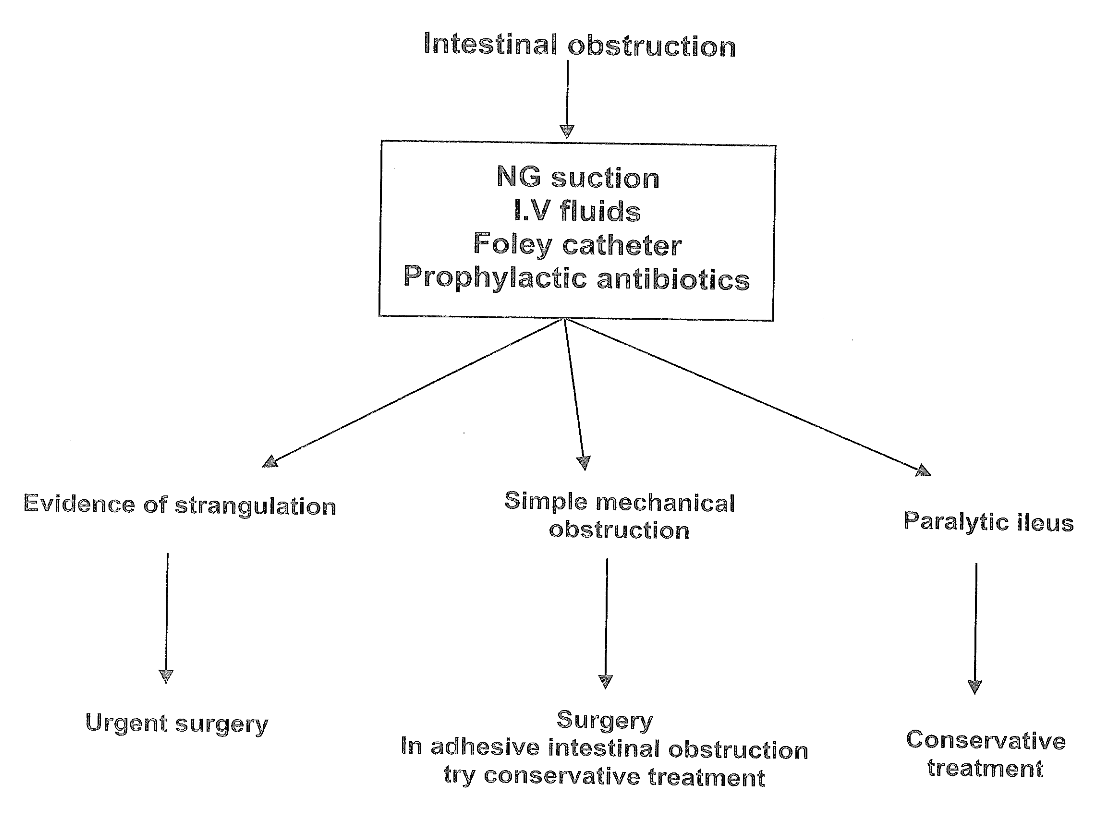
Prevention

1. Prevention and correction of biochemical disturbances. Hypokalaemia is prevented and treated by intravenous potassium administration that is guided by serum level estimation.
2. Gentle handling of the intestine during surgery.
3. For major abdominal surgery, a naso-gastric tube is used to decompress the bowel postoperatively.

Treatment

1. Intravenous replacement of the lost fluids and electrolytes, and naso-gastric suction (drip and suck) are the essentials of treatment.
2. Correction of underlying metabolic abnormalities and hypoproteinaemia.
3. If a postoperative ileus is unduly prolonged, one should think of peritonitis from a leaking intestinal anastomosis and of mechanical obstruction from early fibrinous adhesions. Both conditions necessitate reoperation.
4. The above mentioned measures are usually successful, but occasionally in resistant cases a parasympathomimetic, e.g., prostgimine, may be useful.

Fig. (36.56): is an algorithm for the principles of management of intestinal obstruction.



Rectal prolapse

The protrusion of the rectum through the anus is called rectal prolapse. Two different types of the disease should be distinguished. When the mucosa only prolapses it is termed partial (or mucosal) rectal prolapse. If the whole thickness of the rectal wall is evened to the outside, it is termed complete rectal prolapse.

Aetiology

Partial prolapse

Partial prolapse in children is common and is probably the result of a combination of factors that include:

1. Loss of the curve of sacrum, the anus and rectum thus forming a vertical tube.
2. Loss of weight results in loss of the supporting pararectal and ischiorectal fat.
3. Straining at defaecation due to prolonged diarrhoea, or whooping cough.

Partial rectal prolapse may occur in adults due to:

1. Advanced cases of haemorrhoids.
2. Continuous straining due to urethral stricture.
3. Atony of the sphincter mechanism in the elderly.
4. Iatrogenic injury to a large portion of the anal musculature during a fistula operation.

Complete prolapse

The condition is more frequent in the elderly, particularly females. In Egypt, young males may be more affected due to bilharziasis of the rectum.

The exact pathophysiology of complete rectal prolapse is still unknown. Many theories are suggested:

1. Failure of rectal support by the levators and pelvic fascia due to defective collagen maturation in the pelvic floor. Excessive stretching of these structures during parturition may explain the association of prolapse with childbirth.
2. Rectal prolapse may represent a sliding type of hernia. In favour of this theory is that most patients have a deep rectovesical or rectovaginal extensions of the pouch of Douglas.
3. Abnormal mobility of the mesorectum leading to lack of fixation between the rectum and sacrum
4. Pelvic floor denervation secondary to repeated childbirth.
5. prolapse is due to intussusception which starts at about 10 cm from the anal verge.
6. In Egypt bilharzial proctitis and colitis may be important predisposing factors as they lead to continuous tenesmus and straining at defecation especially if there is bilharzial ulcers and polyps.

Clinical features

Symptoms

- **Prolapse**. In early phases the prolapse appears on straining at defaecation and then reduces spontaneously. Later it may require manual repositioning.
- Mucus discharge, bleeding from the congested mucosa and discomfort are common symptoms.
- A degree of faecal incontinence is a common symptom with complete rectal prolapse, and this may be the distressing symptom that urges the patient to seek medical advice.
- Irreducibility, ulceration, and gangrene are possible complications.

Signs

- The patient is asked to strain in order to demonstrate his/her complaint. Examination in the squatting position is required if the prolapse does not show in the lateral position.
- The anal sphincter tone is also assessed.
- A distinction should be made between the mucosal and the complete forms of rectal prolapse. The differences between partial and complete rectal prolapse are illustrated in Table 36.2.

Table 36.2. Differences between partial and complete rectal prolapse.

Partial prolapse		Complete prolapse
≤ 5 cm	**Length**	> 5 cm
Absent	**Mucosal corrugations**	Often present
Mucosa only	**Thickness**	Whole rectal thickness

Differential diagnosis

1. Prolapsing haemorrhoids.
2. Prolapsing polyp.
3. Prolapsing intussusception.

Treatment

Children

Surgery is rarely indicated as the condition resolves spontaneously with the progress of age.

- Avoidance of constipation and straining.
- Improving the nutritional status.
- Manual reduction by the mother after defaecation followed by strapping of the buttocks to prevent prolapse. The mother is advised to postpone training the child to defecate in the potty.
- Submucous injection of 5% phenol in almond oil as for haemorrhoids, is done in resistant cases.

Adults

Mucosal prolapse in adults. In early cases submucous injection of 5% phenol in almond oil may be successful. If the prolapse is large it is excised in a fashion similar to haemorrhoidectomy.

Complete prolapse in adults requires more sophisticated surgery. The results are clouded by a proportion who get a recurrence and by those who continue to have faecal incontinence. Various surgical procedures include:

1. Rectopexy. Through an abdominal approach the rectum is mobilized, and is pulled upwards. A rectangular piece of polypropylene mesh or ivalon sponge is attached by sutures to the presacral fascia, and is then wrapped and sutured around the back and sides of the rectum. The fibrous tissue initiated by the presence of a foreign material fixes the rectum in its new high position.
2. Excision of the redundant rectum. The operation can be performed either through an abdominal or a perineal approach.
3. Delorme's operation is done through a transanal approach. The excess rectal mucosa above the dentate line is circumferentially excised. Sutures are used to

approximate the upper edge of the remaining mucosa to that at the dentate line, the sutures are made to pass through the rectal muscle, plicating it.

4. Narrowing of the anus (Thiersch's operation) is reserved for the frail patient who is judged unable to stand a major surgery. The prolapse is reduced, and a stainless steel wire or a thick monofilament suture is tunneled subcutaneously around the anus. The suture is then tied to fit snugly around the finger of the surgeon. Though the operation is the simplest, it provides the poorest results.

PEDIATRIC SURGERY

General Principles

Infants and children, in general, show major differences from adults. Their body systems have yet to develop and their full physiological functions and reserves are small. Thus it is easy for a small change to produce organ failure in one system; which may lead to failure of other systems.

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Rate of change. Children are very fragile; a child can be moribund from meningitis within 24 hours or become dehydrated from gastroenteritis to the extent of peripheral circulatory failure in a day.

Temperature regulation. The small baby is easily affected by changes in environmental temperature. This is due to:

- Thinner layer of subcutaneous fat.
- Inadequate vasoconstriction of cutaneous vessels in response to cold.
- Absent or rudimentary shivering and sweating mechanisms
- Incomplete myelination of the CNS including the central heat regulating centre in the hypothalamus. As a result, all premature babies and most neonates will require incubators or at least wrapping of limbs and body with cotton wool to minimize heat loss.

Body fluids and electrolytes. 80% of the full-term baby's weight is water. This drops to 60% in adults. Maintenance of fluid and electrolyte balance is vital in the neonatal period. If oral intake is not feasible IV daily fluids are given.

- Daily fluid maintenance is given in the formula of 10% glucose in 0.18 percent saline. Potassium chloride (20 mmolIL) is added once urine flow is established.
- The fluid requirement of the neonate may be calculated according to the scheme in table 37.1.
- All fluid losses, for example, nasogastric aspirate and ileostomy losses are replaced with normal saline with potassium chloride (20 mmol/L).
- The above are merely guidelines and each case must be assessed individually with repeated electrolyte estimations.

Renal adjustment takes hours to days. As with adults and even more quickly, a baby may develop respiratory or metabolic acidosis or alkalosis. A simple formula that can be used to estimate the amount of bicarbonate needed to infuse in the first instance in the common condition of metabolic acidosis is: Number of mmol **HCO_3 = Base deficit X Wt in Kg/3**. However, proper correction of electrolyte and acid-base imbalance necessitates frequent monitoring of blood values several times daily.

Blood volume. In a neonate, the blood volume is about 80 ml/kg body weight (70 ml/Kg in adults). Thus a 3.5 kg neonate will have 280 ml total blood volume. Three blood stained swabs during surgical operation will contain 30 ml of blood which constitute 10% of the total blood volume. Such loss is significant in neonates but very negligible in adults. Blood transfusion may be resorted to in restricted indications, e.g. blood loss, haemolytic

anaemia or severe anaemia. A general formula of 10 ml/kg of blood or 5 ml/kg of packed RBCS is usually adequate.

Respiratory system. The lungs have sufficient capacity for postnatal survival at about 24 weeks gestation. After delivery, the lung matures rapidly but only about half of the alveoli are present at birth. It is worth remembering that the most energy consuming activity of the neonate is breathing. This explains the rapidity th which respiratory failure develops in babies with pneumonia.

	Maintenance IV fluids/day
Neonate	60 ml/kg to be raised by l0 ml/kg daily up to l00 ml
<10kg	100 ml/kg
> 10 kg	100 ml/kg/ for the first 10 kg + 50 ml for each additional kg of body weight

Hepatic function. The newborn liver is still immature, The most common example of this is the physiological jaundice that is brought about by the underdeveloped glucoronyl transferase system. Many other enzymes and functions are also involved, e.g. albumin, clotting factors and vitamin K. The liver acts also as a carbohydrate store in the form of glycogen. Even the full-term baby can not withstand starvation more than 6 hours without running the risk of hypoglycaemia.

Feeding. Feeding may be by the following methods, breast, bottle, nasogastric tube, gastrostomy, trans-gastric duodenal or jejunal tube or intravenous fluid therapy. If the baby cannot tolerate enteral feeding for more than a week, total parenteral nutrition (TPN) is mandatory.

Central nervous system. Myelination within the central nervous system is poorly developed at birth. The swings in heart and respiratory rate and temperature regulation are examples of the immature negative feedback loops at birth. The process of myelination is not completed until the late teens. The development milestones of childhood are related to the gradual maturation of the cortical connections within the brain.

Pain relief and analgesia. It was commonly stated that infants do not feel pain because of lack of central myelination. There is enough evidence to suggest that babies and small children produce physiological response to pain. It is also important to remember that the blood brain barrier is less efficient until 2 years of age due to incomplete myelination. As a result, fat-soluble drugs will permeate more freely into the cells in the brain in this age group. Thus opiates may produce respiratory depression at low doses. For example, morphine is given to adults in a dose of 0.2 mg/kg 6-8 hourly, but in the small infant 0.1 mg/kg 6-8 hourly is enough.

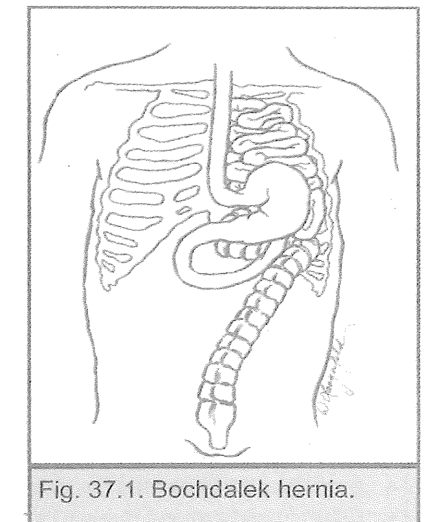


Fig. 37.1. Bochdalek hernia.

Congenital diaphragmatic hernia (CDH):

Any newborn presenting with respiratory distress, in the absence of heart disease, and a scaphoid abdomen should be considered as having congenital diaphragmatic hernia until proved otherwise. The incidence of CDH is 1:4000.

Embryology and aetiology:

Important developmental events:

1. Fusion of the septum transversum and the pleuroperitoneal folds occurs bilaterally during the 8th week of intrauterine life (right side before the left).

2. The intestine returns into the abdomen for rotation and fixation at the 10th week.

Consequences of diaphragm maldevelopment

- If diaphragmatic development is incomplete by the 10th week, the pleuroperitoneal hiatus (foramen of Bochdalek) persists and intestinal loops may herniate into the chest resulting in posterior diaphragmatic hernia. This will result in compression of the lung interfering with its development. As a result, there may be severe hypoplasia of the lung.
- Failure of fusion between the costal and sternal parts of the diaphragm will result in anterior (Morgagni) diaphragmatic hernia.
- Eventration of the diaphragm is a rare condition characterized by severe attenuation of its muscular and fibrous parts. Thus, the diaphragm is very flabby and protrudes into the pleural d'avity.

Types of diaphragmatic hernia

1. Posterior or Bochdalek hernia (Fig. 37.1) constitutes 90% of cases (left side more than right, Lt:Rt = 5:1).
2. Anterior or Morgagni hernia,
3. Hiatal hernia (through the oesophageal hiatus).
4. Eventration of diaphragm (not a true hernia).

Pathology:

The main problems in diaphragmatic hernia are:

1. Ipsilateral lung hypoplasia results in cyanosis and pulmonary hypertension, and leads to right to left shunt.
2. Contralateral lung compression by the abdominal contents.
3. Ischaemia of the herniated abdominal contents.

Clinical features:

Antenatal diagnosis:

- Maternal polyhydramnios raises suspicion.
- Antenatal ultrasound is diagnostic.

After birth:

In severe cases with marked pulmonary hypoplasia symptoms appear immediately after birth and include:

- Therei is respiratory distress, and gasping.
- General examination reeals cyanosis and tachypnea.
- Chest examination reveals that the heart sounds are better heard on the right side in case of the common left sided hernia. Intestinal sounds may be rarely heard on the chest.
- The abdomen is scaphoid.

Mild cases may present late by recurrent chest iinfections or by accidental auscultation of bowel sounds on the chest.

Investigations:

- Chest X-ray reveals gas shadow of the stomach or bowel in the thorax (Fig. 37.2).
- Blood gases to assess the degree of hypoxia.
- CT scan to the chest

Differential diagnosis:

Other causes of neonatal respiratory distress, e.g. congenital heart disease, oesophageal atresia, aspiration, and hyaline membrane disease

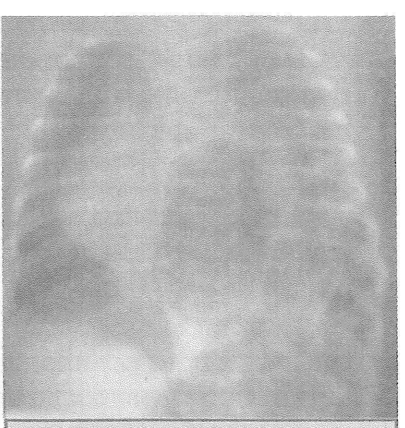


Fig. 37.2. Chest X-ray of a neonate with Bochdalek hernia. Intestinal loops that are filled with gas are seen inside the left side of the chest. The heart is pushed to the right.

Treatment:

Preoperative care (in a paediatric surgical center)

- Naso-gastric tube insertion to deflate the stomach.
- Endo-tracheal tube intubation and ventilatory support to correct the hypercapnea and acidosis.
- IV fluids
- Incubator to keep body temperature.

Operation:

Through an abdominal approach, the herniated abdominal contents are reduced and the defect is closed directly or a mesh is used if the defect is too big.

Post-operative care:

Continued ventilatory support is usually required. Honeymoon period lasts between 2-5 days postoperatively after which pulmonary hypertension develops and causes right to left shunts and eventually death may occur in about 50% of cases.

Modern trends in treatment:

- Intra-uterine surgical correction has limited success.
- ECMO (Extra Corporeal Membranous Oxygenation) is used in some centres to facilitate oxygenation until the lung matures.
- Nitric Oxide and toulazoline are used in some centres as they induce pulmonary vasodilatation.

Oesophageal atresia

A newborn presenting with repeated accumulation of frothy saliva should be considered to have oesophageal atresia until proved otherwise.

The Incidence is 1:4000. Males are affected more than females and the condition is more common in white than black races.

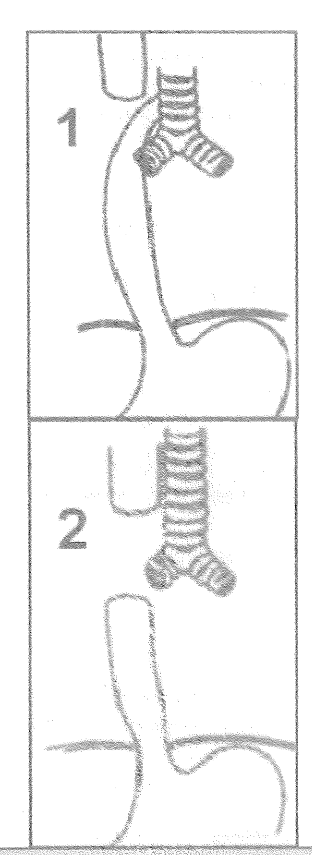


Fig. 37.3. Oesophageal atresia.
1. With tracheo-oesophageal fistula (common).
2. Without fistula (rare).

Aetiology

The condition is due to a defect in the division of the proximal fore-gut into ventral tracheobronchial tube and a dorsal oesophageal tube.

Pathology

Types: (Fig. 37.3)

1. The commonest type of oesophageal atresia is proximal blind atresia with distal tracheo-oesophageal fistula (87%).
2. Rare types include atresia without fistula (7%).

Complications:

1. As a result of proximal oesophageal atresia, the saliva and milk (if the baby was fed) will fill the upper pouch then trickle into the trachea and mouth causing bronchopneumonia and frothy saliva.
2. As a result of the distal tracheo-oesophageal fistula, the acidic gastric juice will regurgitate through the trachea into the lungs causing acid pneumonia which if persists is fatal.
3. Failure of feeding will lead to dehydration and hypoglycaemia.

Association anomalies:

The acronym "VACTERL" helps to remember a group of congenital anomalies that are commonly associated.

Vertebral (spina bifida with its various types, chapter 57).
Anorectal.
Cardiac (chapter 28).
Tracheal.
Esophageal.
Renal (30%, chapter 43).
Limb anomalies.

Clinical features:

Antenatal diagnosis:

- Maternal polyhydramnios may be present as in any case of antenatal intestinal obstruction.
- Antenatal ultrasound may show a dilated upper pouch of the oesophagus.

At birth:

1. Repeated accumulation of frothy saliva after proper resuscitation.
2. Attacks of chocking and cyanosis.
3. Pneumonia develops rapidly if the condition was not diagnosed before or immediately after birth.
4. Abdominal examination reveals abdominal distension with air if the fistula is large enough. If oesophageal atresia is suspected oral feeding is strictly prohibited until the condition is excluded.

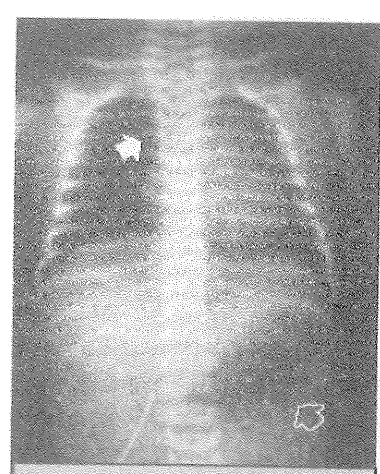


Fig. 37.4. Plain X-ray in a case of tracheo-oesophageal fistula. White arrow indicates the site of arrest of a nasogastric tube. Transparent arrow points at excess gastric air that indicates the presence of a fistula.

Investigations:

1. Catheter test. Insert a radioopaque catheter into the nose down to the oesophagus. If the catheter is arrested at 10cm, this denotes oesophageal atresia.
2. Plain x-ray of the chest and abdomen after inserting a nasogastric tube. This will confirm the presence of atresia of the oesophagus. The presence of a gastric air bubble will confirm the presence of a fistula (Fig. 37.4).
3. Other investigations to evaluate associated anomalies, e.g., renal ultrasound or echo-cardiography.
4. Blood gases to assess the need for assisted ventilation.

Treatment:

Early diagnosis before starting oral feeding is crucial to avoid pneumonia. The treatment is by urgent surgery in a paediatric surgery centre.

Preoperative care:

- Oral feeding is prohibited.
- Continuous low pressure suction of saliva.
- The infant is kept in an incubator.
- IV. fluids and antibiotics.
- Transfer to paediatric surgery centre.

Operation:

- Atresia with a fistula. Right thoracotomy, ligation of the fistula, and primary anastomosis of oesophageal ends should be tried.
- Atresia without fistula. Usually there is a long gap which makes immediate anastomosis difficult. A gastrostomy is, therefore, done at birth. Further surgery to restore oesophageal continuity is to be planned 6 months later.

Congenital hypertrophic pyloric stenosis (CHPS)

Any neonate presenting with projectile, non bile stained vomiting not responding to antiemetics and associated with hunger should be considered to have CRPS until proved otherwise.

Incidence:

- 1/400 neonates.
- M:F=4:1
- More in first born babies.
- Familial predisposition. The condition is more frequent in babies whose mothers had pyloric stenosis as neonates.

Aetiology:

No definite cause has been identified. The following factors, however, may be implicated.

- Improper innervation of the pyloric sphincter.
- Reduced nitric oxide synthesis.
- Increased growth hormone level.

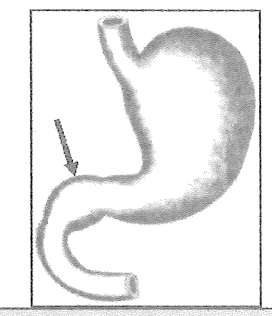


Fig. 37.5. Hypertrophied pyloric sphincter.

Pathology:

- At birth the pylorus is normal, but within 2-3 weeks, there is gradual progressive hypertrophy of the circular pyloric muscles (Fig. 37.5).
- This results in gradual occlusion of the gastric outlet. The baby continues to suckle but the milk cannot pass beyond the stomach leading to projectile vomiting.
- Recurrent, frequent vomiting results in loss of fluids, acid, chloride and salt. Thus blood chemistry often shows hypochloremia, hyponatremia, hypokalaemia and alkalosis.
- The stomach contracts trying to push its contents against the pylonc resistance, this explains the visible gastric penistalsis usually seen over the abdominal wall.

Clinical features:

Symptoms:

- Vomiting is the main feature. Vomiting is projectile, and non- bilious. It usually starts 2-3 weeks after birth. It does not respond to antiemetics. Sometimes the vomiting may be delayed. After vomiting the infant is very hungry and cries for its feeds.

- Firm stools.
- Loss of weight.
- In 10% of cases the infant has haematemesis due to gastritis or oesophagitis.

Signs:

- General examination, Dehydration is present in neglected cases.
- Abdominal examination:
    - The hypertrophied pyloric muscle is usually felt as an olive-like mass, Feeling this mass is diagnostic of the condition. The olive mass is felt in the transpylonic plane to the right of the right rectus muscle, better when the stomach is empty.
    - Test feed. The mother is asked to feed the baby, and then the examiner inspects the abdomen for visible peristalsis passing from left to right and palpates the abdomen for the olive like pyloric mass which is felt in the epigastric region on the undersurface of the right lobe of the liver.

Differential diagnosis:

- Other causes of neonatal vomiting.
- Gastroenteritis.
- Gastro-oesophageal reflux.
- Feeding problems.
- Intestinal obstruction.

Fig. 37.6. Barium meal showing elongated narrow pyloric canal

Investigations:

- **Imaging** is required only if the olive tumour is not palpable.
    - **Ultrasound**. This is the imaging modality of choice. CHPS is diagnosed if the thickness of the pyloric muscle is more than 4 mm.
    - Barium meal is performed if the facilities and experience in paediatric ultrasound are not available. The radiologtcal features are:
        - Dilated stomach.
        - Exaggerated peristalsis.
        - The pyloric canal is elongated and narrow (Fig. 37.6).
        - Delayed gastric emptying.
- **Laboratory investigations** (serum electrolytes): These are crucial to assess electrolyte deficits as these babies usually suffer from hypochloraemia, hyponatraemia, hypokalaemia and alkalosis.

Treatment:

Treatment is surgical. It requires proper preoperative preparation.

Preoperative care:

- Correction of dehydration and electrolyte deficits by IV fluids before surgery.
- Stop oral feeding.

It usually takes 24 hours to rehydrate the baby to be fit for surgery.

Operation. Ramstedt pyloromyotomy (Fig. 37.7):

- General anaesthesia.
- The abdomen is entered through a small transverse right upper abdominal incision.

- The hertrophied pylorus is grasped between the index and the thumb and a myotomy is performed until the mucosa of the pylorus bulges.
- If the mucous membrane is injured, the injury should be sutured and reinforced by an omental patch.

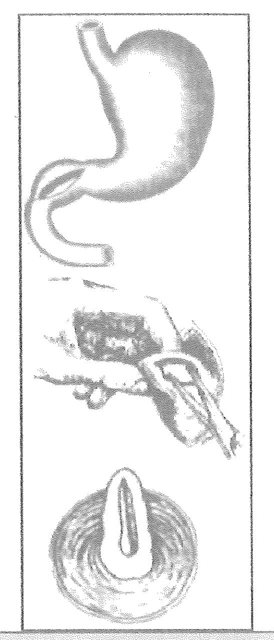


Fig. 37.7. Ramstedt's operation.

Postoperative care:

Oral feeding can be resumed after a few hours.

Prognosis. The results of this operation are excellent and the mortality should be around zero.

Intestinal obstruction in neonates, infants and children:

Main causes:

- **Duodenal obstruction:**
    1. Atresia.
    2. Malrotation with bands or volvulus (volvulus neonatorum).
    3. Annular pancreas.

1. **Jejunal and Heal obstruction:**
    1. Volvulus neonatorum.
    2. Atresia.
    3. Meconium ileus.
    4. Peritoneal bands.
    5. Strangulated inguinal hernia.
    6. Intussusception (chapter 36).
    7. Postoperative peritoneal adhesions, e.g., following appendicectomy.
2. **Large bowel obstruction:**
    1. Hirschsprung's disease.
    2. Anorectal anomalies.
    3. Meconium plug syndrome.
3. Necrotizing enterocolitis
4. Adynamic intestinal obstruction, e.g., due to hypokalemia or pneumonia.

Early detection:

1. Ante-natal. Polyhydramnios raises the suspicion of high gut obstruction.
2. During routine assessment of a neonate, that has just been delivered, a tube is passed down the oesophagus for aspiration of secretions and to exclude oesophageal atresia. The tube is further passed down to the stomach. A neonate with bilious gastric aspirate more than 25ml should be considered to have intestinal obstruction until proved otherwise,
3. Any child presenting with persistent, bile-stained vomiting should be considered to have intestinal obstruction until proved otherwise. Waiting for this stage, however, carries the risk of aspiration pneumonia.

Clinical features:

As for adults, the cardinal signs of intestinal obstruction are 2V, 2C, 2D, i.e.,
2 "V"s Vomiting and Visible peristalsis.
2 "C's Colics and Constipation (absolute).
2 "D's Distension and Dehydration.

1. Vomiting is the cardinal symptom of all types of intestinal obstruction. It may be early in cases of proximal intestinal obstruction, or late in distal intestinal obstruction. Greenish or brown faeculent vomiting is diagnostic of intestinal obstruction.
2. Visible peristalsis usually indicates mechanical obstruction; They are:
    a. In the epigastrium if obstruction is gastric or duodenal.
    b. Central abdominal in small intestinal obstruction.
    c. Peripheral abdominal in colonic obstruction.
3. Colics are manifested in babies by crying and withdrawing of legs.
4. Constipation. Failure to pass meconium or stools for more than 24 hours usually indicates intestinal obstruction in infants and neonates.
5. Distension follows the same pattern as visible peristalsis i.e. It is:
    a. Epigastric in gastric or duodenal obstruction.
    b. Central abdominal in small intestinal obstruction.
    c. Peripheral abdominal in colonic obstruction. However, if distal intestinal obstruction is presenting late, there may be diffuse severe abdominal distension.
6. Dehydration

Management

First aid

1. **Insertion of 3 tubes**
    a. Nasogastric tube.
    b. IV. access.
    c. Urinary catheter or urinary bag.
2. Fluids and drugs
    a. Ringer's lactate solution 10-20 ml/kg over the next hour according to the degree of dehydration.
    b. Antibiotics to cover both aerobic and anaerobic organisms, e.g. peflicillin, gentamycin and metronidazole.
3. Electrolytes and acid base fine adjustments according to blood chemistry.

Confirm diagnosis

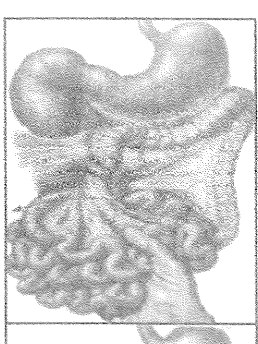
1. Plain abdominal x-ray: This is the first and most important investigation. It may reveal the presence of distended bowel loops ad the presence of more than three fluid levels. The gas pattern usually helps to identify the cause of many problems.
    a. Double bubble in duodenal obstruction.
    b. Free air in peritoneum in intestinal perforation
    c. Calcification in intra-uterine perforation.
    d. Ground glass in meconium ileus.
    e. Intra-mural gas in necrotizing enterocolitis.
2. Barium meal may be needed to diagnose incomplete intestinal obstruction and mairotation.
3. Gastrografin enema is particularly helpful in Hirschsprung's disease and meconium ileus. In the latter it may also be therapeutic.
4. Rectal biopsy may be needed for suspected Hirschsprung's disease (colonic aganglionosis). Definitive treatment

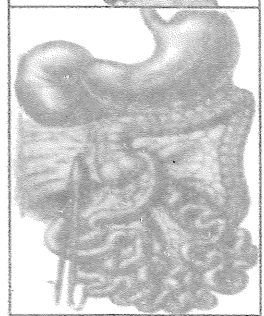
Individual causes of paediatric intestinal obstruction

Volvulus neonatorum

- The condition is caused by incomplete rotation of the midgut that should occur during intra-uterine life (chapter 36).

- The baby is born with a free midgut having a narrow-based mesentery, and is, therefore, liable to twist around its mesenteric axis. The result is strangulation obstruction of the whole midgut.
- In addition, there is usually a fibrous band ([add's band) that extends from below the liver to the left to the caecum which lies in the epigastrium. This can obstruct the duodenum.
- Urgent surgery is needed to untwist the gut, to divide the band and to broaden the base of its mesentery by further displacement of the caecum to the left (Fig. 37.8).





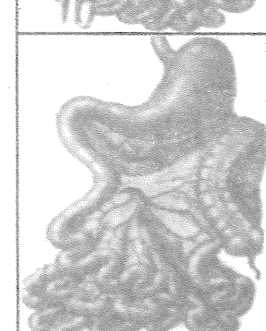


Fig. 37.8. Volvulus neonatorum. Untwisting, division of band of Ladd and displacement of the caecum to the left.

Jejunal and ileal atresia

- Jejunal and ileal atresia are due to obstruction of mesenteric arteries in utero.
- The atretic segment of bowel may be a cord like structure (Fig. 37.9) or may be completely absent.
- In jejunal atresia there is early bilious vomiting but distension is lacking.
- In ileal atresia abdominal distension is marked while vomiting may be delayed.
- In both conditions some meconium may be evacuated.
- Plain abdominal X-ray reveals distended bowel loops with fluid levels.
- Urgent surgery is required. Resection of the atretic segment is done with bowel anastomosis.

Meconium Ileus:

- There is obstruction of ileum in a neonate due to thick inspissated meconium.
- The condition occUrs in 10% of patients with cystic fibrosis (mucoviscidosis).
- Gastrografin enema may be diagnostic as well as therapeutic. Gastrografin absorbs water that may dissolve the thick meconium.
- Ileostomy may be needed and may be life saving.

Peritoneal bands:

Peritoneal bands, e.g., band of [add, or bands in relation to Meckel's diverticulum may compress the lumen or precipitate volvulus.

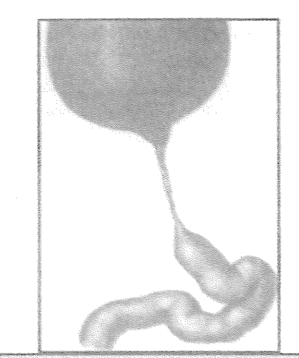


Fig. 37.9. Ileal atresia with proximal dilatation.

Strangulated inguinal hernia:

Strangulated inguinal hernia is a common cause of partial or complete intestinal obstruction during the first 6 months of life. Urgent surgery is required.

Hirschsprung's disease (aganglionic megacolon):

Any newborn presenting with delayed passage of meconium for more than 24 hours should be considered as having Hirsch- sprung's disease until proved otherwise.

Incidence is 1:5000

- M:F 4:1.
- There is familial incidence in 6% of cases.

Aetiology:

- The disease is due to absence of ganglia in the submucous and intermucular plexuses. The disease starts in the upper part of the anal canal and extends proximally to the recto sigmoid area in 80% of cases (typical case) or it may involve variable lengths of the colon or even the whole colon.
- The affected segment of bowel is spastic and narrow. The normally ganglionic colon proximal to it is markedly dilated (Fig. 37.10).

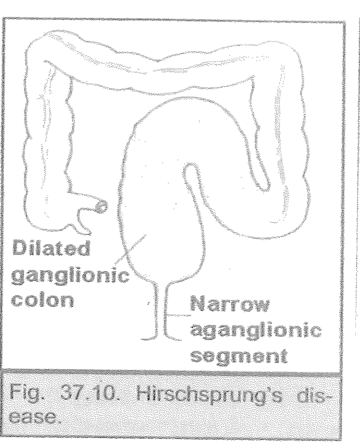


Fig. 37.10. Hirschsprung's disease.



Fig. 37.11. Barium enema of congenital megacolon showing a narrow aganglionic distal segment (arrows) and marked proximal colon dilatation.

Clinical features:

- The problem starts since birth. There is delayed passage of meconium. The mother notices that the infant is suffering from chronic constipation. Defecation occurs every few days and only after insertion of a suppository or doing an enema.
- Progressive abdominal distension develops.
- Sometimes, chronic intestinal obstruction culminates into acute obstruction with severe abdominal distension, absolute constipation and there may be vomiting.
- A severe complication called obstructive enterocolitis is liable to occur. During the affack the patient develops pyrexia, abdominal distension and diarrhoea.
- Delayed growth and development.
- Digital rectal examination reveals an empty rectum and when the finger is removed, it is followed by a sudden gush of gases or stools.

Investigations:

- Plain abdominal x-ray reveals large bowel obstruction.
- Barium enema needs to be done without preparation and a small amount of barium is administered. It usually shows the narrowed aganglionic segment, followed by a dilated normal ganglionic bowel (Fig. 37.11). Its diagnostic accuracy is 80%.
- Rectal biopsy. Previously full thickness rectal biopsy was recommended, but nowadays only a small segment of the mucosa and submucosa is taken by a special forceps. Ganglion cells in the submucous layer are absent.
- Anorectal manometry reveals failure of relaxation of the anal phincters in response to rectal distension.

Treatment:

- Patients presenting by intestinal obstruction
  - Initial conservative treatment by nasogastric tube suction, IV. fluids and repeated colonic washouts with saline.
  - If the obstructiion is relieved, prepare the patient for later elective surgery.
  - If the obstruction is not relieved perform urgent colostomy.
- Patients without obstruction require elective surgery.
  - Surgery is ideally performed at the age of 6-9 months.

- Definitive surgery for Hirschsprung's disease entails com plet excision of the aganglionic segment of the bowel up to 4 inches above the funnel and then anastomosis of the nor ma colon to the lower half of the anal canal.

Anorectal malformations:

Types:

- High anomalies where the rectum ends above the level of the pelvic floor and it is either a blind pounch or more com monl there is a fistulous connection to the bladder or pos terio urethra (Fig. 37.12) in males or to the vagina in fe males.
- Low anomalies where the rectum has passed through the pelvic floor and usually opens into an ectopic site anterior to the normal anus position. A fistulous connection may pass to the vestibule in females or to the perianal skin in males (Fig. 37.13).

Fig. 37.12. High imperforate anus.

Incidence 1:5000.

- In high anomalies M:F2:1.
- In low anomalies M:F = 1:2.

Associated anomalies:

VACTERL. These associated defects are often serious and adversely alter the prognosis for the infant. They are more common in the high than in the low types.

Clinical features:

Examination of the perineum:

- Look for the presence of anus, its size and site.
- Look for the presence of an anal dimple.
- If there is an impulse on crying at the site of the anus, it denotes a low anomaly.
- In ectopic anus, there is a subcutaneous fistulous tract full of meconium.
- Meconium at the tip of the penis denotes a listula to the bladder or urethra
- If a thermometer can be introduced into the anus for more than one cm, this excludes rectal atresia.

Fig. 37.13. Low imporforate anus. In this neonate a fistula opens by a tiny opening at the base of scrotum.

Abdominal examination:

There may be evidence of intestinal obstruction.

General examination:

To exclude associated congenital anomalies.

Investigations:

- Plain X-ray in the inverted position (Invertogram). The idea of this radiological test is that 24 hours after delivery, enough gas has traveled along the colon to reach the distal end of the bowel. The baby is put in an inverted position for a few minutes and a coin or a small barium paste is put at the possible site of the anus. The distance between the radio-opaque mark and the distal gas shadow is measured. If the

distance is more than 1 cm, this denotes high anomaly, while if less than 1 cm, a low anomaly is diagnosed.

- Urine is examined for the presence of meconium, if present; this denotes a fistulous communication to the bladder.

Surgical Treatment:

High anomalies are treated with.staged surgery. First stage is a temporary colostomy. Second stage is abdomino-ano-rectal pull-through. Third stage is closure of colostomy. Results are not usually satisfactory because continence may be weak. Low anomalies require only local perineal surgery. Results are excellent in the majority of patients.



Invertogram showing a high anorectal anomaly

Meconium plug syndrome:

This term is used to describe distal colonic obstruction associated with narrow left colon and thick plug of meconium in the distal colon. The baby is otherwise normal and gastrograf'in enema is usually curative.

Necrotizing enterocolitis (NEC):

This is the most serious and frequent gastrointestinal disorder of predominantly premature infants with a median onset of 10 days after birth. The incidence is increasing in view of the therapeutic advances in neonatal intensive care that allowed many more premature babies to survive.

It is characterized by necrosis, ulceration and sloughing of intestinal mucosa which frequently progresses to full-thickness necrosis and perforation. Gas-forming bacteria in the intestinal wall may lead to pneumatosis (air in the intestinal wall), a finding which is noted in plain abdominal radiographs in the supine or lateral positions.

Contrast studies are hazardous and may lead to perforation.

Treatment includes cessation of feedings, orogastric suction, systemic antibiotics and correction of hypoxia, hypovolaemia, acidosis and electrolyte disturbances.

The only absolute indication for operation is intestinal perforation.

At Laparotomy, the necrotic bowel is resected and the proximal bowel is made into a stoma as a primary anastomosis may not be safe. Late sequelae include short bowel syndrome due to extensive resections or intestinal strictures as a result of the healing process.

Childhood tumours:

Tumours in children show major differences from adult tumours. They are less common, show different cell types, and totally different biological behaviour. In general, tumours in children show better response to combination of surgery, chemotherapy and radiation. As a result, childhood tumours have better prognosis than adults tumours.

Common tumours in children (in order of frequency)

1. Acute lymphocytic leukaemia.
2. Brain tumours.
3. Neuroblastoma.
4. Nephroblastoma.
5. Hepatoblastoma.

6. Rhabdomyosarcoma.
7. Sacrococcygeal teratoma.

Common tumours in different age groups:

Neonatal period:

- Ovarian cystic lesions.
- Metastastasing neuroblastoma (curiously with excellent prognosis).
- Sacrococcygeal teratoma.

0-2 years:

- Nephroblastoma (Wilms' tumour).
- Neuroblastoma.
- Hepatoblastoma.

2-6 years

- Rhabdomyosarcoma of the bladder or prostate.
- Ovarian teratoma.
- Malignant lymphoma.

Clinical features of abdominal tumours in children:

- Abdominal mass. This is the commonest presentation especially in Wilms' tumour and neuroblastoma.
- Some tumours present by systemic manifestations as fever, anorexia and failure to thrive. A common example is neuroblastoma.
- Some neoplasms present by neurological symptoms as paresis or incontinence, e.g. neuroblastoma and rhabdomyosarcoma.
- Pain may occur due to metastases or torsion of an ovarian cyst.

APPENDIX

CHAPTER CONTENTS

Surgical anatomy

The vermiform appendix is a blind-ending hoflow muscular tube that arises from the caecum at the confluence of the taeniae coil. It averages 7.5 to 10 cm in length.

Position (Fig. 38.1)

- The relation of the appendix to the caecum is variable. The commonest position of the appendix is retrocaecal (74%), and the second common is the pelvic position (21%). Less common are the paracaecal (2%), the subcaecal (1.5%), pre-ileal (1%) and least of all positions is the post-ileal (0.5%) appendix.
- Furthermore, the location of the caecum is not always in the right iliac fossa. The ascending colon may be so short that the caecum is subhepatic, or may be so long that the caecum may lie in the pelvis. Pregnancy also alters the position of these organs as the enlarging uterus progressively displaces the caecum and the appendix up towards the right hypochondrium.
- The length of the mesoappendix is variable, and in some cases it is nonexistent, whereas the appendix lies beneath the serosa of the caecum and is called a subserous appendix.

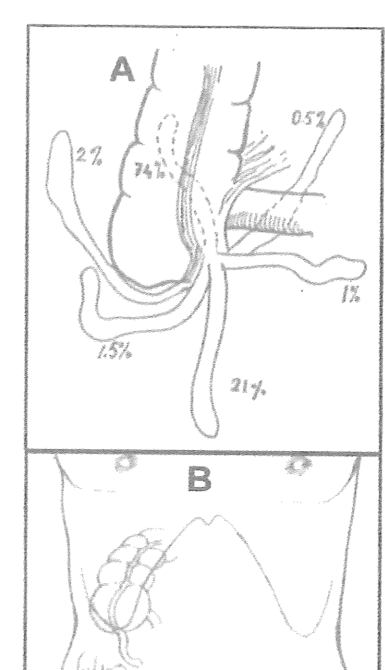


Fig. 38.1. Position of the appendix.
A. In relation to caecum.
B. Variation in position of caecum.

Blood supply

- **Arterial supply**. The appendix has a profuse blood supply via one or two appendicular arteries arising from the ileocolic artery. The arteries run in the mesoappendix, but they are closely applied to the wall distally, therefore, with acute appendicitis, thrombosis and consequent gangrene are likely to affect the distal part of the appendix.
- **Venous drainage**. Likewise the organ is drained by tributaries of the ileocolic vein, to the superior mesenteric, and then the portal vein.

Histology

- **Mucosa.** The appendicular mucosa is similar to the colonic mucosa being formed of columnar epithelium with fewer goblet cells, Crypts from the mucosa are present but are shorter and fewer than in the colon. At the base of the crypts are many Kultchitsky cells (or argentaffin cells).
- **Submucosa**. The lumen of the appendix is irregular and narrow due to the abundance of lyrnphoid follicles in the submucosa. This lymphoid tissue progressively atrophies with age.

- **The most common cause of emergency abdominal surgery is acute appendicitis.**
- **The risk of perforation is 25% after 24 hours from onset of pain and rises to 50% after 36 hours.**

- **Musculosa**. The musculosa is thin and is formed of two complete layers of circular and longitudinal muscles.
- **Serosa**. The appendix is completely covered by peritoneum.

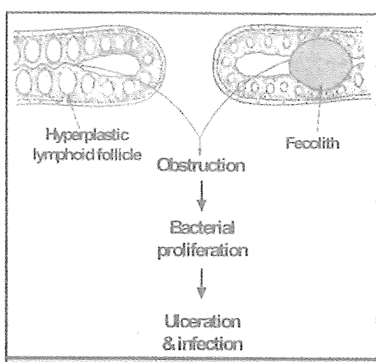


Fig. 38.2. Sequence of events in obstructive appendicitis.

Acute appendicitis

Prevalence

- Appendicitis is the most common acute abdominal surgical emergency.
- In Western countries, 16% of the population undergo appendicectomy.
- It is most prevalent in the second and third decades of life, after which the risk declines with age. Since the appendix of the infant is wide-mouthed and well drained, and since in the elderly the umen is almost obliterated, and the wall contains little lymphoid tissue, appendicitis in the two extremes of life is relatively rare.
- Racial differences do exist. Appendicitis is more common in Western countries and in urban than rural areas, which is probably related to low fibre diet.

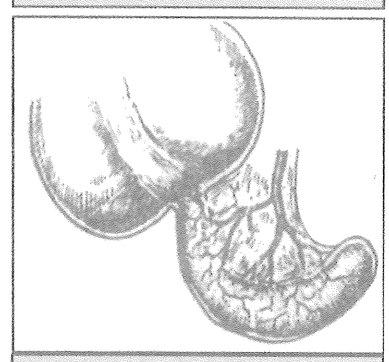


Fig. 38.3. Suppurative appendicitis.

Pathology

Gross types

- In about two thirds of cases inflammation is initiated by obstruction of the lumen, an entity that is termed obstructive acute appendicitis. Obstruction is usually caused by a faecolith (inspissated stool), or by swelling of the lymphoid tissue in response to viral infection (Fig. 38.2). Less common causes are kinking by adhesions, intestinal parasites, e.g. pin worms, and tumours of the appendix or the caecum. Distention of the obstructed appendix promotes overgrowth of the resident bacterial flora and subsequent invasion of the mucosa, starting the process of appendicitis which is rather rapid, and tends to be suppurative (Fig. 38.3) or gangrenous (Fig. 38.4).
- Non-obstructive acute appendicitis accounts for one third of cases. Inflammation usually starts in the mucosa and tends to remain there (catarrhal inflammation). It may, however, spread but at a slower rate and is less likely to perforate.



Fig. 38.4. Gangrenous appendicitis.

Course

In untreated cases the course of the disease is variable.

1. The acutely inflamed appendix may resolve, but if so, a further attack (or attacks) is likely.
2. Persistent obstruction, particularly with a faecolith, tends to produce gangrene and perforation. It occurs either at the tip where the appendicular vessels are very close to the wall or are even intramural, or at the

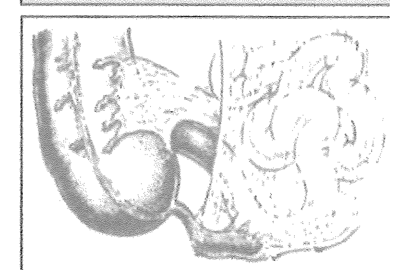


Fig. 38.5. Start of an appendix mass formation.

site of impaction of the faecolith. The incidence of perforation is high in the very young below 5 years, and in the elderly. Perforation is followed by generalized, or localized peritonitis, depending on the severity of inflammation on one side, and the efficiency of the body defense on the other.

3. When the inflammatory process is slower, the body defense has the time to wall off the inflamed appendix by adhesions to the neathy intestine and the omentum (Fig. 38.5), and within 3 to 5 days of the start of pain an appendicular mass (phlegmon) forms in the right iliac fossa, or in the pelvis.
4. Perforation of the appendix within an appendicular mass produces an appendicular abscess.
5. Portal pyaemia (pylephlebitis) is a rare complication. It results from suppurative thrombophlebitis of the portal venous system. Chills, high fever, low-grade jaundice, and, later, hepatic abscesses are the hallmarks of this grave condition.

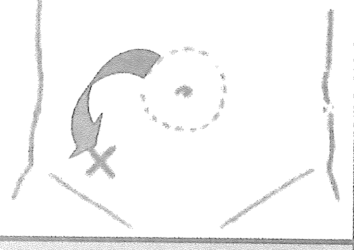


Fig. 38.6. Acute pain that shifts.

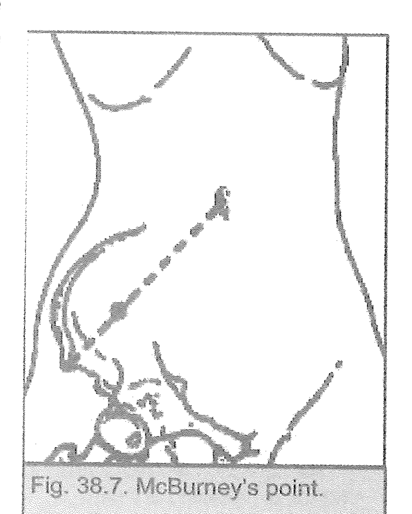


Fig. 38.7. McBurney's point.

Clinical featuers

Proper history taking and physical examination are the corner stone of diagnosis.

Symptoms

1. Acute pain that shifts is the presenting symptom. Classically it starts in the periumbilical region and is ill localized. Distention of the appendix produces this visceral pain that is felt in this area because the appendix is a part of the midgut. Within 6-10 hours when the inflammation proceeds to irritate the parietal peritoneum, the pain shifts to the right iliac fossa (Fig. 38.6) and becomes sharper and more localized (somatic pain). The pain is aggravated by movement or cough.
2. Anorexia and nausea are nearly always present. Vomiting occurs in about three fourths of the patients, is not usually repeated, but is always preceded by the pain. If vomiting precedes the abdominal pain, one should think of another diagnosis.
3. Constipation is usual.

Physical examination

Physical findings are affected by the stage of the disease and the position of the appendix.

1. Temperature and pulse are only slightly elevated eally in the disease. Higher elevations indicate a complication, such as perforation. Appendicitis does not start by a rigor or a temperature above 40°C.
2. If the patient is asked to cough, the pain becomes sharp and well localized to the site of the appendix. He is then asked to point to the area of maximum pain.
3. Localized tenderness (anJ probably rebound

Acute appendicitis diagnostic facts

- Diagnosis is essentially clinical.
- Acute pain that shifts (Fig. 38.)
- Anorexia is almost always present. If the patient feels hungry and can eat, the diagnosis of acute appendicitis is in serious doubt.
- Pain precedes vomiting. If vomiting comes first consider the diagnosis of gastroenteritis.
- Localsied tenderness, usually in right iliac fossa. Sometimes point of tenderness is low in the pelvis or high in right hypochondrium (according to site of appendix). See Fig. 38.1.
- A normal leucocyte count does not exclude appendicitis, but a high count with shift to the left supports the clinical diagnosis.

tenderness) is present in the right iliac fossa, usually, but not always, over the McBumey's point which is at the junction of the inner two thirds and the outer third of a line extending from the umbilicus to the anterior superior iliac spine.

4. Rigidity indicates perforation.
5. Restricted abdominal wall movement with respiration indicates peritonitis.
6. If the patient presents on the third or fourth day of the attack, a tender appendicular mass may be felt in the right iliac fossa.
7. Pelvic examination either vaginal or rectal is essential to exclude gynaecological problems. It also reveals tenderness or a mass in case of pelvic appendicitis.
8. Special signs, if present, assist in the diagnosis.
    a. Rovsing's sign is pain felt in the right lower quadrant on deep palpation of the left lower quadrant.
    b. Psoas sign is pain on extension of the right thigh. It is caused by psoas spasm as a result of irritation by retrocaecal appendicitis.
    c. Obturator sign is pain on internal rotation of the flexed right thigh that is caused by irritation of the obturator internus by pelvic appendicitis.

Atypical clinical features

1. Inflammation of an appendix that points deeply in the pelvis induces tenesmus from rectal irritation, and dysuria from bladder irritation. Abdominal signs are minimal and tenderness is elicited on pelvic examination.
2. Pain and tenderness are higher than the McBurney's point when the caecum is high. With a subhepatic appendix, the condition is commonly misdiagnosed as cholecystitis.
3. With a long retrocaecal appendix pain is felt in the right loin. Abdominal examination reveals minimal tenderness or guarding.
4. Diarrhoea may occur when the ileum is irritated by an inflamed retro-ileal appendix.
5. Appendicitis in infants and young children is more serious. Below the age of 3 years, 80% of patients will have perforated appendicitis due to the following reasons:
    a. Difficulty of examination of an infant or young child.
    b. The greater omentum is not well developed and so localization is difficult.
    c. There is prominent vomiting and the case may wrongly be diagnosed as gastritis or enteritis.
6. Acute appendicitis in the eldeily produces little tenderness and rigidity, though perforation is relatively common. Eliciting these signs is even more difficult if the patient is obese. The danger of late diagnosis of a perforated appendix is further aggravated by the natural weakness of the immune system that ensues with age.
7. Appendicitis with pregnancy. Usually the site of pain is displaced upwards as pregnancy progresses. Also the localization by the omentum is less efficient and the condition is often misdiagnosed as pyelitis. If the diagnosis and operation are delayed until perforation, there is a higher chance of abortion or premature labour (50% of cases). While if the patient is operated upon before perforation, premature labour occurs only in 30% of cases.

Differential diagnosis

Many diseases or groups of diseases can mimic acute appendicitis.

- **Group 1**. Thoracic problems

Right sided pneumonia or right basal pleurisy can mimic appendicitis but usually the abdomen is not rigid, there is minimal tenderness and there are marked chest symptoms.

- **Group 2**. Upper abdominal problems
  1. **Perforated peptic ulcer**. Usually there is a history of chronic dyspepsia. When perforation occurs there is a sudden onset of severe abdominal pain and tenderness and mild shock. Then there follows a period of mild improvement (lucid interval) where peritoneal fluid dilutes the gastric contents in the peritoneum. Later follows a steadily increasing peritonitis stage. There is gradually increasing pain, tendemess, and progressing rigidity. The rigidity may be very marked in the right iliac fossa because gastric or duodenal contents flow along the right paracolic gutter to the right iliac fossa. However, tenderness and rigidity are more marked in the right hypochondrium and epigastric regions. A plain X-ray usually shows gas under the diaphragm.
  2. **Acute cholecystitis**. Here the signs and symptoms may resemble appendicitis but they are definitely more marked in the right hypochondrium rather than the right iliac fossa. The history is more suggestive and the vomiting is more severe. Ultrasonography will clinch the diagnosis.
  3. **Cyclic vomiting of children**. This may be a real problem in childhood but the temperature is normal, rigidity never occurs and the leucocytic count is normal.
- **Group 3**. Lower abdominal problems
  1. Non specific mesenteric lymphadenitis. The commmonest organism is Yersinia. The patient is usually a child with pain which comes in attacks. The tenderness shifts with changing of position. It is difficult to be sure about the diagnosis.
  2. Iliac adenitis. Usually there is a demonstrable cause for the adenitis and the site of maximum tenderness is much lower near the iliac vessels.
  3. Cancer of the caecum. This is more likely to be misdiagnosed as appendicular mass rather than appendicitis.
  4. Regional ileitis. In its acute form it may be indistinguishable from appendicitis unless a vague doughing mass can be felt. A definite history of intestinal trouble, with repeated diarrhoea and bleeding is very suggestive.
  5. Meckel's diverticulitis, This may closely resemble appendicitis. It is usually not diagnosed except after exploration.
  6. Gastroenteritis.

Group 4. Pelvic problems

Problems of the female genital system may resemble appendicitis to a great extent.

1. Ruptured graffian follicle (mittelschmerz) may produce mid-cycle ovulation pain.
2. Ruptured ectopic pregnancy.
3. Tubal abortion.
4. Twisted ovarian cyst.
5. Pelvic inflammatory disease (PID), e.g. salpingitis, pyosalpinx, and tubo-ovarian abscess.
6. Red degeneration in a fibroid.

Conditions 1-3 can be highly suspected if a careful detailed menstrual history is taken especially as regards the exact date of last menstrual period and any delay in the cycle.

All conditions 1-6 differ from appendicitis in that:

- Pain and tenderness are often bilateral and more in the suprapubic area.
- Conditions associated with bleeding may have mild hypothermia (temperature 36.2-36.6) tachycardia, pallor and may be low blood pressure.
- High temperature may be present in salpingitis which may be bilateral.
- P-V examination is very informative.

- Sonography is of great diagnostic importance.

- **Group 5**. Urological problems
  1. Right renal ache or ureteric colic (right ureter stone).
  2. Right pyelonephritis.
     - Often start with a rigor and temperature 39-40°C.
     - Pain is more in the loin rather than iliac fossa.
     - Pain radiates to the upper thigh, testicle and suprapubic area.
     - Often associated with dysuria and/or pyuria.

> **Common differential diagnoses of acute appendicitis.**
> - **Right tubo-ovarian causes (see text).**
> - **Mesenteric adenitis (Yersinia).**
> - **Right ureter stone.**
> - **Gastroenteritis**
> - **Meckel's diverticulitis.**

- **Group 6. Neurological problems**

The pain which precedes the eruption of herpes zoster affecting 10th, 11th and 12th thoracic nerves. The pain is very similar in site and distribution to appendicitis but there is no tenderness or rigidity and the pain does not shift. Eruptions may appear 36-48 hours later.

Differential diagnosis of an appendicular mass

A mass in the right iliac fossa is most likely an appendix mass. Other possibilities include:

1. Carcinoma of the caecum. The condition is a chronic one and is likely to be associated with anaemia and weakness. The mass is usually hard and not tender.
2. Crohn's disease
3. Hyperplastic ileocaecal tuberculosis.
4. Right iliac lymphadenitis.

Investigations

Requested investigations should be urgently done. The first two of the following investigations areroutinely done for all suspected acute appendicitis cases.

1. **Leucocyte count**. A moderate leucocytosis (10,000-16,000/uL) with a predominance of neutrophils is present in 8090% of cases. However, a normal white cell count does not rule out appendicitis.
2. **Urine analysis may show few red blood cells**. The presence of a large number of RBC5, or of pus cells raises the suspicion of a ureteric calculus, or of urinary tract infection respectively.
3. Pregnancy test if ectopic pregnancy is suspected.
4. **Ultrasound examination**
   - Abdominal ultrasound may diagnose appendicitis in some cases where the appendix appears distended and noncompressible. A fecolith may also be detected. In children the inflamed appendix is usually >10 mm in diameter.
   - More important is the use of ultrasound for exclusion of other diseases that simulate acute appendicitis. Examples are:
     - Females with suspected gynaecological problems. Transvaginal ultrasonography is particularly useful in these cases.
     - Suspected right ureter stones where back pressure changes show in right kidney.
5. **Laparoscopy**. It is the policy is some centres to perform laparoscopy for females in the child-bearing period, who present with lower right abdominal pain. The procedure is done under general anaesthesia. It may reveal a nonsurgical problem, e.g. pelvic inflammatory disease or mid-menstrual ovulation pain, and the patient is spared the trouble of surgery. On the other hand a surgical problem can be dealt with in the same session either by open surgery, or if facilities and experience exist,

by laparoscopic surgery. Tubal pregnancy, ovarian cysts, and acute appendicitis, for example, can be managed laparoscopically

Treatment

A patient with acute appendicitis presents with one of the following situations:

1. Uncomplicated acute appendicitis.
2. Appendicular mass.
3. Appendicular abscess.
4. Perforated appendicitis with generalized peritonitis.

Uncomplicated acute appendicitis

Treatment is "urgent appendicectomy". The operation should not be delayed especially in children, elderly persons, pregnant females and diabetic patients.

Appendicular mass

Urgent appendicectorny is not recommended because:

1. An appendicular mass represents success of the body to isolate the danger. It is better left undisturbed.
2. Appendicectomy is technically difficult and carries the hazard of injuring the adherent intestine leading to peritonitis or faecal fistula.

Initial conservative treatment is adopted. This treatment is named Ochsner-Sherren regimen after the two surgeons who described it.

1. Rest in bed for the first few days.
2. Intravenous fluids are administered until nausea subsides and the patient is able to start oral feeding. Oral intake starts with fluids, followed by gradual introduction of solid meals.
3. Antibiotics. A combination of ampicillin, aminoglycoside and metronidazole can cover most of the pathogenicorganisms. Antibiotics are administered IV at first then orally.
4. An analgesic may be prescribed for the pain.
5. Meticulous observations are made for the temperature, pulse, pain, vomiting, degree of tenderness and rigidity and for the size of the mass.

In 80-90% of patients the appendicular mass will resolve under conservative treatment. The patient is discharged and is scheduled for interval appendicectomy after 3 months. If the operation is not performed, there is a high risk of a further attack of acute appendicitis.

Appendicular abscess

This means accumulation of pus inside an appendicular mass with deterioration of the general and local conditions.

It is suspected when there is an unduly rising pulse rate and pyrexia. Persistent vomiting, and persistent or rising pain after the first few hours of treatment, increasing rigidity and an enlarging mass are also criteria to abandon conservative treatment. Ultrasound examination further confirms the formation of fluid in the mass. In such a case urgent drainage of pus is needed either by open surgery, or by ultrasound-guided percutaneous aspiration which spares the patient the trouble of anaesthesia and surgery. The route of open drainage depends on the location of the abscess. If it was felt in the right iliac fossa, the operation is done through an iliac muscle cutting incision, trying to reach the abscess cavity through an extraperitoneal approach whenever possible. All loculi are opened, the pus drained, and a rubber drain is left in place for a few days. No attempt

is made to remove the adherent friable appendix at this stage. A pelvic abscess, on the other hand, is drained transanally through the anterior wall of the rectum.

The patient is discharged within days and is scheduled for "interval appendicectomy" after three to six months. This time interval allows the inflammatory mass to resolve completely. If the operation is not performed, there is a high risk of a further attack of acute appendicitis.

Perforated appendicitis with generalized peritonitis

Urgent surgery is required. The perforated appendix should be removed and peritoneal toilet is performed.

Chronic appendicitis

Chronic, or more correctly recurrent subacute appendicitis, is a cause of recurrent attacks of abdominal pain and dyspepsia. In most of the cases it is difficult to prove beyond doubt that chronic appendicitis is the cause of pain. Even when pain and tenderness are centred around the right lower abdomen, it is advisable to exclude other causes of chronic pain before removing the appendix. Amoebic colitis, irritable bowel syndrome, chronic calcular cholecystitis, and Crohns disease should be bom in mind. The treatment of chronic appendicitis is appendicectomy.

Neoplasms of the appendix

Carcinoid Tumour (Argentaffinoma)

This is the commonest tumour of the appendix. The appendix is also the commonest site in the gut (65%) to be affected by carcinoid tumour. The ileum is affected in 25%.

Pathology

The tumour arises from Kulchitsky cells in the depth of the mucosal pits. The behaviour of the tumour is usually benign, but a minority invade (5%) and metastasize to the liver. The distinction between benign and malignant tumours depends mainly on the biological behaviour. Tumours less than one cm are usually benign while those more than 2 cm are almost always malignant and metastasize, The latter may produce the rare carcinoid syndrome because of their production of serotonin.

Clinical features

1. Commonly asymptomatic.
2. Carcinoid tumour is sometimes an accidental histological diagnosis in an appendix that has been removed because of chronic or acute appendicitis. Examination of the cut section shows that the tumour appears to arise from the muscle layer and has a characteristic bright yellow colour caused by its lipoid content.
3. Carcinoid syndrome is rare and is produced only by malignant carcinoid tumours that metastasize to the liver. The syndrome includes one or more of the following; flushing with attacks of cyanosis, diarrhoea with borborygmi, bronchospasm, and pulmonary stenosis. The diagnosis of the carcinoid syndrome is confirmed by finding high urinary concentrations of the serotonin metabolite 5-hydroxyindoleacetic acid (5-HIPA).

Treatment

- For a tumour that is less than 2 cm, and is still confined to the appendix, appendicectomy is a sufficient treatment.
- For larger lesions, right hemicolectomy is required.

Adenocarcinoma

Adenocarcinoma of the appendix is rare. It may present as acute appendicitis. The tumour behaves as carcinoma of the colon. If it is confined to the appendix and the regional lymph nodes, the treatment is right hemicolectomy.

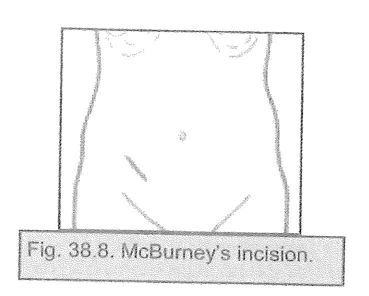


Fig. 38.8. McBurney's incision.

Appendicectomy

Indications

1. Acute appendicitis is the commonest indication. The operation is an urgent one.
2. For appendicular mass and abscess. appendicectomy is performed 3-6 months after resolution.
3. Chronic appendicitis.
4. Carcinoid tumours that are confined to the appendix and are smaller than 2 cm.

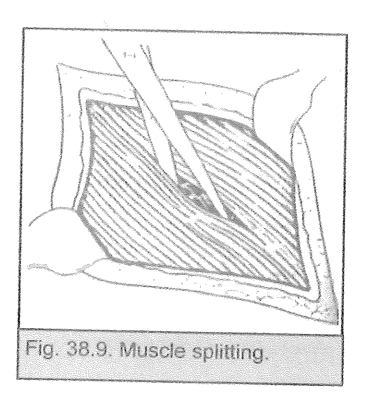


Fig. 38.9. Muscle splitting.

Normal appendix at operation

- If at operation the appendix looks normal, the surgeon should check the terminal ileum (Meckel's diverticulum, mesenteric adenitis and Crohn's disease), and the right Fallopian tube and ovary.
- Up to 20% negative appendicectomies is acceptable. It is better to take out some normal appendices than to miss a case of acute appendicitis that goes on to rupture.

Steps

1. The patient should be fasting for a few hours before the operation.
2. **Prophylactic parenteral antibiotics are used. They are** started one hour before the operation and may be continued after the operation for a variable time depending on the severity of appendicitis and the presence of peritonitis. For uncomplicated cases one preoperative dose and another given after six hours are sufficient; the so called perioperative antibiotic prophylaxis. It is customary to use a combination of an agent against anaerobes (e.g. metronidazole, or clindamycin) and another against gram negative bacilli (e.g. an aminoglycoside).
3. The operation is done under general or spinal anaesthesia. The patient lies in the supine position.
4. Examination under anaesthesia. A previously impalpable appendicular mass may be felt under anaesthesia. In such case, the operation is postponed.
5. Incision. For most patients, the best is a gridiron (McBurney's) incision (Fig. 38.8) located over the area of maximal tenderness. This is usually, but not always, the McBurney's point. If the diagnosis is highly doubtful, an exploratory lower right paramedian incision is preferable. The gridiron incision is an oblique one 2-3 inches long. The external oblique is incised parallel to its direction, and the internal oblique and transversus abdominis muscles are split also along the direction of their fibres (Fig. 38.9). The peritoneum is then opened. In some cases upward and lateral extension of the incision is needed to improve access. This is done by cutting through the internal and transversus abdominis muscles, an incision that is known after Rutherford Morison.
6. If pus or serous fluid is present in the peritoneal cavity, a sample is taken for culture and sensitivity. The pus is then evacuated.

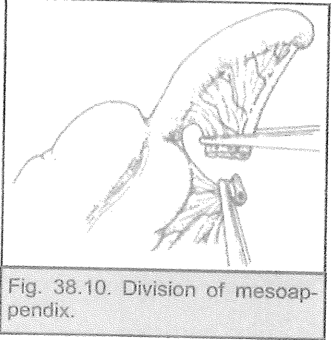


Fig. 38.10. Division of mesoappendix.

7. Exploration of the pelvis and the terminal ileum is done to exclude gynaecologic problems and Meckel's diverticulitis for patients in whom the appendix is not apparently inflamed.
8. Finding the appendix. The caecum is located and the appendix is found at the confluence of the taeniae coli.
9. The mesoappendix containing the appendicular vessels is doubly ligated at its base and is then divided near the appendix (Fig. 38.10). Whenever the mesoappendix is thickly laden with fat, or is involved in inflammatory oedema, a ligature tightened around its whole thickness is likely to be insecure. In such cases it is advisable to ligate a smaller piece of the mesentery at a time (piecemeal ligation).
10. The base of the appendix is ligated, and a clamp is applied distal to the ligature. The appendix is then divided with the scalpel just below the clamp (Fig. 38.11).
11. The appendix stump may be embedded by tightening a purse string suture that has been passed in the serosa and muscle of the caecum around the stump, and placed half an inch from it (Fig. 38.12). While tightening, the appendix stump is pushed down to be buried. The added safety of burying the appendix stump with a purse string suture is disputed by many surgeons who omit this step without ill effects. Whenever the wall of the caecum at the base of the appendix is oedematus, this suture should be avoided since it is prone to cut through while it is tightened.
12. In an adherent retrocaecal appendix that is devoid of a mesentery, retrograde appendicectomy is easier. First the appendix base is ligated and divided to separate it from the caecum. The blood supply is then secured by clamps and ligatures bit by bit, working from the base to the tip.

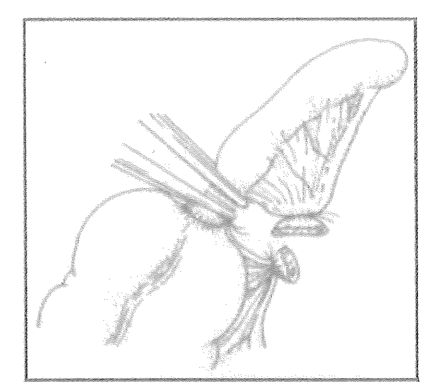


Fig. 38.11. Division of the appendix base.

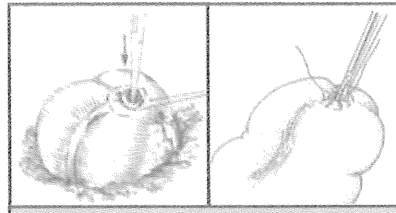


Fig. 38.12. Embedding the appendix stump by a purse string suture.

13. In cases of peritonitis, care is taken to clean the peritoneum of pus. An intraperitoneal drain, brought out through a separate stab, may be inserted,
14. Wound closure. The wound is closed in layers. If the wound is severely contaminated by pus, it is very likely to get infected. In such cases it is advised to adopt the policy of delayed primary closure where the skin is left open, to be closed by adhesive tape or sutures 4-5 days later.

Postoperative care

- The patient is given regular analgesics and IV fluids.
- He is kept fasting till the return of intestinal sounds when gradual oral intake is started.
- The skin stitches are removed around the 7th postoperative day.

Complications

The operation is a relatively safe one. The mortality for uncomplicated cases is 0.1% or less, but rises to about 3.5% in cases with perforation. Postoperative complications include:

Complications common to all surgical procedure

- Bleeding from a slipped mesoappendix ligature.
- Chest infection.
- Wound infection. This is the commonest postoperative complication of acute appendicitis, and its incidence is much higher in perforated cases.
- Deep vein thrombosis.
- Distension due to paralytic ileus or early fibrinous adhesions.
- As any intraperitoneal surgery fibrous adhesions may form and lead to ate intestinal obstruction, or female infertility.
- Incisional hernia. A grid iron incision is unlikely to develop an incisional hernia. This is because it is a muscle splitting incision that produces no muscle damage, and the fibres of the three abdominal muscles that are oriented in different directions prevent herniation with straining. However, a wound that has been extended by muscle cuffing, or one that has become infected, is prone to develop an incisional hernia. Furthermore, a high muscle cut may injure the ilio-inguinal nerve causing paralysis of the conjoint muscle producing a paralytic direct inguinal hernia.

Complicafions that are specific to appendicectomy

- Peritonitis.
- Iliac or pelvic abscess.
- Faecal fistula.

Laparoscopic appendicectomy

This relatively new technique is evolving as a substitute for the formal operation. The main advantage is its exploratory potential, whereas the whole peritoneal cavity is inspected. Less morbidity, and hospital stay, together with faster recovery are also possible assets. This operation, however, is not yet as widely accepted and practiced as laparoscopic cholecystectomy.

THE ANAL CANAL

Diseases of the anal canal constitute some o fthe most frequently seen cses in medical and surgical practice.

CHAPTER CONTENTS

Surgical Anatomy

The anal canal is the terminal part of the gastro-intestinal tract.

Development

The upper fourth of the anal canal is derived from the distal part of the hind gut, while its lower part is derived from the proctodaeum which is an invagination of the ectoderm. The two separately developed parts meet at the dentate (pectinate) line which can be seen on proctoscopy.

Course and relations (Fig. 39.1)

- The anal canal starts at the level of the pelvic floor, ends at the anal opening, and measures 3-4 cm in length.
- As the rectum passes through the pelvic floor (levator ani muscle), it ends by turning backwards at a right angle forming the anal canal.
- It continues downwards and backwards, surrounded by three concentric cylinders of muscles. These are the two well distinct internal and external sphincters, with the thin longitudinal muscle layer between them.
- The anal canal is cushioned on either side by the fat-filled ischiorectal fossa.

Anal canal inner lining

- The mucous membrane above the dentate line is lined by cubical epithelium which changes gradually upwards until it becomes transformed into columnar epithelium at the rectum.
- Below the dentate line the anal canal is lined by modified skin lacking hair follicles or sweat glands and is called anoderm.
- Minute anal glands open on anal crypts that are circumferentially arranged and are situated at the junction of the two embryologically distinct parts. The appearance of these crypts as they join each other forms the dentate line.
- Above the dentate line the mucous membrane is thrown into longitudinal folds called columns of Morgagni.

Blood supply

Arterial supply

- The superior haemorrhoidal (superior rectal) artery is the direct continuation of the inferior mesenteric. It supplies the upper part of the anal canal by three terminal branches, two to the right and one to the left of the middle line.
- The middle haemorrhoidal (middle rectal) artery is a branch of the internal iliac. It arises on either side and passes medially along the lateral ligaments.
- The inferior haemorrhoidal (inferior rectal) artery arises from the internal pudendal branch of internal iliac. As the internal pudendal artery passes through the pudendal canal it gives off the inferior rectal artery which crosses the ischiorectal fossa medially

to supply the lower part of the anal canal. There is a free anastomosis between the three arteries forming a rich submucous and intramural plexuses.

The **venous drainage** runs parallel to the arterial supply. The superior rectal veins drain into the portal circulation through the inferior mesenteric vein, while the middle and inferior rectal veins drain into the systemic circulation through the internal iliac vein. Though the anal canal is a site of porto-systemic anastomosis, the incidence of piles in cases of portal hypertension is equal to that of normal population.

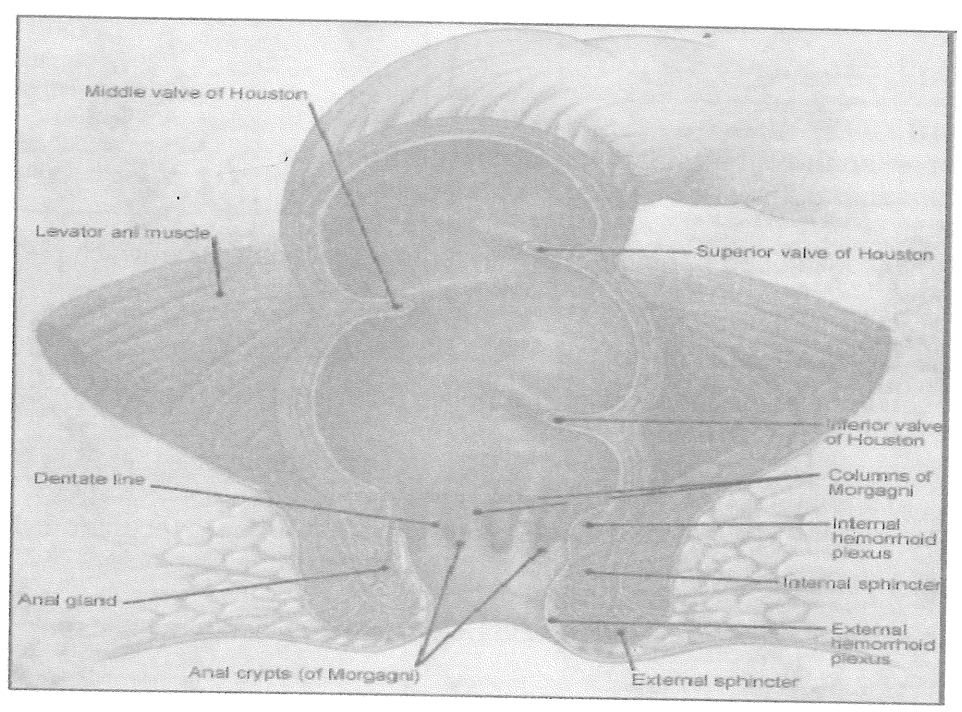


Fig. 39.1. Anatomy of anal canal. The dashed line separates the rectum from the anal canal

Lymphatic drainage

- The upper smaller part of the anal canal, above the dentate line, drains into the superior rectal lymph nodes and then to the pre-aortic nodes.
- The main lower part drains into the superficial and then the deep inguinal nodes on both sides.

Dentate line

The dentate line is an important surgical landmark (Table 39.1).

Table 39.1. Importance of dentate line

	Above dentate line	**Below dentate line**
Development	Hindgut	Proctodaeum
Lining epithelium	Low columnar cells	Skin formed of stratified squamous epithelium that is devoid of sebaceous glands and hair
Appearance	Pink	Parchment-like
Venous drainage	Portal	Systemic
Nerve supply	Visceral, not sensitive to pain	Somatic, sensitive to pain
Lymphatic drainage	Upwards along superior rectal then to pre-aortic nodes	Lateral to inguinal nodes

Anal sphincters

1. The internal sphincter is the thickened continuation of the circular muscle coat of the rectum. It starts where the rectum passes through the pelvic diaphragm and surrounds the anal canal for a distance of about 2.5 cm. The internal sphincter is about 2.5 mm in thickness, and as it is a visceral muscle, its colour is whitish. Spasm of this muscle plays a major role in the pathology of anal fissure and perianal suppuration.
2. The longitudinal muscle is also a visceral (involuntary) muscle which is the continuation of the longitudinal coat of the rectum, mingled with some fibres of the puborectalis muscle. It fans through the lowest fibres of the internal sphincter to be attached to the anal and perianal skin. These lowest fibres are termed the corrugator cutis ani muscle as they are responsible for corrugation of the perianal skin.
3. The external sphincter is a somatic (voluntary) muscle that forms the outermost muscular cylinder. It was formerly described as having three parts, but is now considered to be one continuous cylinder. The muscle is innervated through the pudendal nerve (S2,3,4), The muscle has a pink colour. It is attached posteriorly to the coccyx, and is inserted anteriorly in the midperineal point in the male, and fuses with the sphincter vaginae in females.
4. The anorectal ring surrounds the anorectal junction. It is composed of the joining of the puborectalis part of the levator ani, the upper parts of the internal and external sphincters, and the longitudinal muscle layer. The integrity of this ring is of paramount importance in anal continence, its injury results in faecal incontinence.

Anal physiology

Physiology of continence

The factors that maintain continence to stools and flatus are:

1. **Anal sphincters**. The internal anal sphincter is the major determinant of continence, contributing as much as 80% of the resting pressure within the anal canal. As it is under autonomic control, the internal sphincter is inhibited by the parasympathetic and is stimulated by the sympathetic nervous system. It principally controls the continence to flatus. Only 20% of the resting pressure is due to the external sphincter, which is a striated muscle supplied by somatic nerves. Division of this sphincter, e.g. during fistula surgery, results in a minor functional disability.
2. **Puborectalis and anorectal angle**. The puborectalis muscle forms a sling that pulls the anorectal junction forwards to maintain its right angle (Fig. 39.2). Electromyography revealed a continuous basal resting activity that is controlled by spinal reflexes with no conscious effort (even during sleep). Loss of spinal reflexes in spinal cord injury or damage of the puborectalis produces faecal incontinence. If the intraabdominal pressure is raised by coughing or lifting heavy objects, the anterior rectal wall compresses onto the upper anal canal and occludes its lumen.
3. **Sensation**. Faeces in the rectum produce awareness of filling which contributes to continence. The stretch receptors for this sensation are located in the levator ani muscle and external anal sphincter. The mucosal lining of the upper part of anal canal contains receptors that permit discrimination between flatus and faeces.
4. **Neuromuscular reflexes** in the pelvic floor and external sphincter. There is a spinal reflex between the rectus muscle on one hand, and the pelvic floor and extemal sphincter on the other. When muscles of the abdominal wall contract during coughing or any other form of straining, they raise the intraabdominal pressure. Anal sphincters and pelvic floor muscles contract, in response, to prevent stress faecal incontinence.

5. **Folds of Houston**. Manometry suggests that these folds of rectal mucosal membrane may have a minor role in the maintenance of continence.
6. **Anal cushions** formed by the terminal branches of the superior haemorrhoidal vessels.

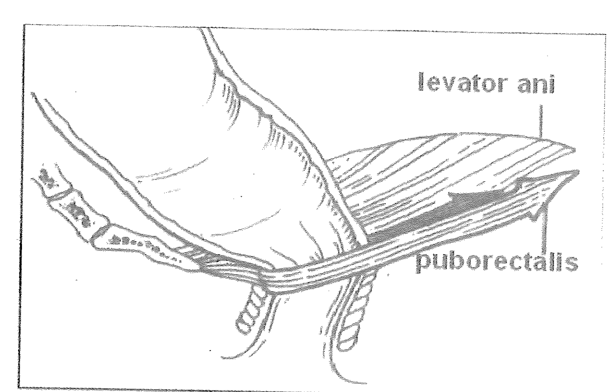


Fig. 39.2. Lateral view that shows the puborectalis pulling the anorectal junction forwards and making a right angle between the rectum and anal canal.

Mechanism of defecation

1. In response to rectal distension by stools or flatus, the internal sphincter relaxes allowing a small sample of these rectal contents to enter the upper anal canal.
2. The sensitive epithelium in this area can discern its type whether it being flatus, liquid, or solid stools (sampling reflex).
3. If the circumstances are convenient, the subject relaxes the puborectalis thus widening the anorectal angle, and also relaxes the external sphincter. Straining will expel the rectal contents to the outside.
4. If the subject does not wish to evacuate the rectum, the puborectalis and the external sphincter contract to push the sample of luminal contents back up.

Pilonidal sinus

Pilonidal sinus is not an anal disease, but is described in this chapter because of its closeness and the possible misdiagnosis as an anal fistula. It is a common minor condition of the skin overlying the sacrum. There is a subcutaneous granulating cavity originally described as a nest (Latin-nidus) containing hair (Latin-pilus) and connected to the skin by midline openings (Latin-sinus).

Aetiology

The disease typically affects young adult males with dense, dark, strong hair. Females are also affected but to a lesser extent. The condition seldom develops after the age of 30 years.

The exact aetiology is not known. There are two possible theories:

- **Congenital theory** claims that congenital dermal inclusions, overlying the lower sacral region, get infected and present after puberty.
- **Acquired theory** is more acceptable, and assumes that loose hairs from the head or back gravitate to the skin over the sacrum and coccyx. There, in the presence of pressure from sitting, a rolling movement of the buttocks, sweat, and poor hygiene, the hairs are drawn through the skin to accumulate in the subcutaneous tissue with debris and organisms. The following facts favour the acquired theory:
    1. Pilonidal sinus may occur in other areas, e.g. in the umbilicus or in the webs of fingers of barbers.

2. Microscopic examination shows only loose hairs in the cavity but there are no hair follicles.
3. The tip of the hair is always directed inward.
4. Liability to repeated recurrences.

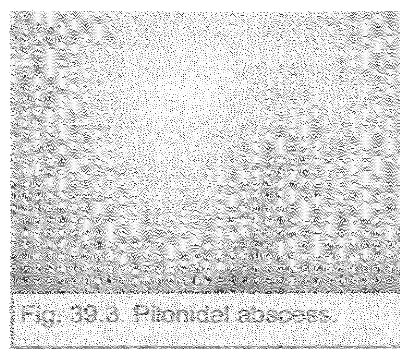


Fig. 39.3. Pilonidal abscess.

Pathology

- Originally, there is a cavity that contains loose hairs, lined by granulation tissue, and lies in the subcutaneous space overlying the lower sacrum. The cavity opens on the skin in the middle line by one or more openings, their tracks being partially epithelialized.
- Intermittently, aerobes and anaerobes proliferate causing an abscess which may empty through the midline openings or point and open laterally and inferiorly producing secondary sinuses. Abscess formation has a tendency to recurrence.

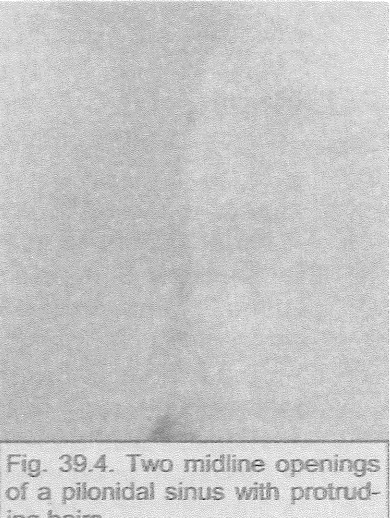


Fig. 39.4. Two midline openings of a pilonidal sinus with protruding hairs.



Clinical features

1. Patients may be asymptomatic.
2. More usually, they present with local discomfort, discharge, or acute abscess formation (Fig. 39.3). Pilonidal disease tends to recur if improperly treated.
3. The discharging midline and lateral openings are seen, sometimes with loose hairs pouting out of them (Fig. 39.4).

Differential diagnosis

1. Perianal abscess.
2. Anal fistula.

Treatment

Pilonidal abscess is initially treated by incision and drainage of pus. Loose hairs are removed, and the wound is left open, packed, and is allowed to heal by granulation tissue formation. If a sinus recurs it is formally treated on an elective basis.

Pilonidal sinus. Different options are available:

- Laying out the cavity and side tracks, and curettage. Phenol cauterization may be applied to the tracks. The resulting wound is left open, packed and is allowed to heal by secondary intention.
- Localized excision of the cavity and side tracks. The wound may be left open to granulate or is closed by sutures to hasten healing.

Wide wedge excision down to the sacrum should no longer be practiced as it is considered to be an unnecessarily extensive operation with very long healing time. After any of the above operations, the patient is advised to keep the area clean and to shave it regularly in order to reduce the possibility of recurrence.

Anal fissure

This is an elongated ulcer which occurs in the lower half of the anal canal, always in the midline. In 90% of cases it lies in the midline posteriorly, while in the remaining 10% the fissure is anterior.

Aetiology

Constipation. It is suggested that the passage of a hard faecal bolus impinges on the posterior wall of the anal canal below the dentate line. The epithelial lining of this area is less supported by muscular tissue and so it is stretched and gives way. The higher percentage of anterior fissures in females can be explained by lack of support to the anal lining due to relaxation or tearing of the perineum during delivery. Rarely fissures may occur due to Crohn's disease.

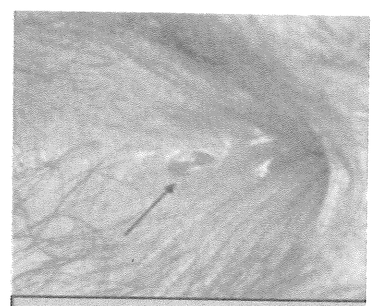


Fig. 39.5. Acute posterior anal fissure. The patient lies in left lateral position during the examination.

Pathology

An anal fissure may occur either in an acute or chronic form.

- An acute fissure is a superficial tear in the lower half of the anal canal in the midline, usually posteriorly. Pain caused by the fissure leads to persistent spasm of the internal sphincter muscle, which prevents its healing.
- Chronic fissure. If the patient is not treated efficiently, the fissure will proceed to the chronic stage where secondary pathological changes occur.
    - The margins become indurated.
    - Fibrosis occurs in the underlying internal sphincter muscle leading to narrowing of the anal canal.
    - An anal papilla may develop at the upper end of the fissure.
    - An oedmatous tag of a skin may form at the lower end of the fissure and is called (sentinel pile).
    - Anal suppuration may develop in relation to the fissure leading to a perianal abscess, which may burst leading to a subcutaneous anal fistula.

Hypertrophied papilla
Dentate line
Fissure
Sentinel pile
Anal verge

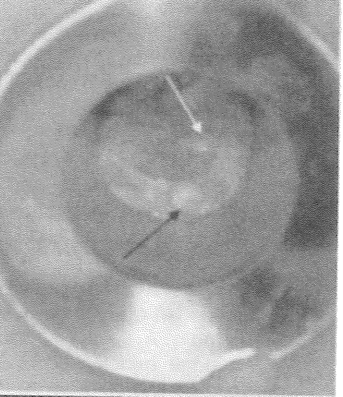


Fig. 39.6. Chronic anal fissure. In the lower picture the proctoscope is inserted under general anaesthesia. For an anal fissure proctoscopy is too painful to be done in the awake patient.
- White arrow: hypertrophied anal papilla.
- Black arrow: sentinel pile.
- A posterior chronic anal fissure lies between them.

Clinical features

Symptoms

1. Pain is the main symptom. Pain is sharp, agonizing, starts during defecation and may continue for a few hours after the act, the patient gets some relief until the next defecation. In subacute cases the patient may get some remission for a few days or weeks but usually the pain recurs.
2. Constipation. The severe pain of the fissure may compel the patient to postpone defecation. This, in turn, will lead to more constipation and a vicious circle is established.

3. Bleeding only a slight streak of blood on the surface of the stools may be present (compare with haemorrhoids).
4. Slight anal discharge may be present. If an abscess forms and bursts there will be purulent discharge.
5. Reflex symptoms are commonly present. Burning micturition, dysmenorrhoea and pain along the thighs may occur.

Examination

- In acute anal fissure, inspection of the anal verge reveals a tightly contracted, puckered anus. If the two gluteal folds are gently pulled laterally, a small tear will be seen in the midline posteriorly (Fig. 39.5). It is very painful and inadvisable to do digital rectal examination at this stage.
- A chronic fissure is seen and induration of the edges is felt. The latter can be done after application of an anaesthetic gel for a few minutes. An anal papilla or a sentinel pile may be present (Fig. 39.6).

Differential diagnosis

1. Early carcinoma of the anus.
2. Fissures due to Crohn's disease. These are usually multiple, occur at any site and are not very painful. They are not indurated.
3. Tuberculous ulcers have undermined, cyanotic edges.
4. Anal chancre presents as a painful ulcer.

In any patient with atypical presentation of anal fissure, histopathological examination of the excised specimen should be performed.

Differential diagnosis of painful anal conditions

1. Anal fissure.
2. Perianal suppuration.
3. Prolapsed strangulated piles.
4. Acute perianal haematoma.
5. Carcinoma of the anus.
6. Proctalgia fugax (idiopathic)

Treatment

Acute fissure

The main aim of treatment is to relieve the persistent spasm of the internal sphincter muscle to give a chance for the fissure to heal. Treatment is **essentially medical** but surgery is sometimes needed.

1. High-fibre diet and some laxatives are prescribed to ensure bulky soft stools.
2. A local anaesthetic ointment as 2% or 5% lignocaine is introduced gently.
3. Recently glyceryl trinitrate ointment 0.2% or bethanecol ointment 1% has been successfully used.
4. Suitable doses of analgesics are prescribed.
5. Frequent warm water baths have a soothing effect.
6. If all the previous measures fail to relieve pain within a few days, the best policy is to do internal sphincterotomy under general anaesthesia.

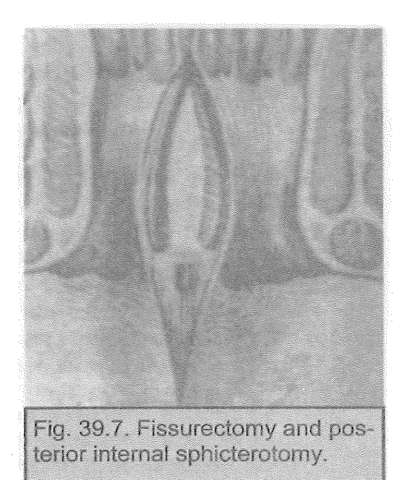


Fig. 39.7. Fissurectomy and posterior internal sphicterotomy.

Chronic fissure

Treatment is surgical

- If the fissure is not very chronic, a lateral internal sphincterotomy operation is very successful. The internal sphincter is divided at the 3 o'clock position. Relief of the spasm of the internal sphincter will allow healing. The operation can be done by the closed or open method.

- If the fissure is heavily fibrosed with a sentinel pile, the best procedure is to do fissurectomy and posterior internal sphincterotomy (Fig. 39.7). A triangular segment with its apex upwards and including the fissure, anal papilla and sentinel pile is excised. The excision includes the fibrosed part of the internal sphincter muscle as well.

Haemorrhoids (piles)

In Greek, haima means blood while rhoos means flowing. In Latin, pila mean a ball. So the accurate terminology should be haemorrhoids if the main complaint is bleeding per rectum or piles if the main problem is prolapse.

Morbid anatomy

Normally the terminal branches of the superior haemorrhoidal vessels form a sort of a vascular plexus beneath the epithelial lining of the anal canal called the "anal cushions". These cushions are usually arranged at the 3,7,11 o'clock positions around the circumference of anal canal. Anal cushions play an important role in the normal continence mechanism. Congestion, enlargement and prolapse of these cushions constitute haemorrhoids,

- Affection of the internal haemorrhoidal plexus which lies above the dentate line produces internal haemorrhoids (Fig. 39.8). They are covered by mucous membrane and are bright red or purple in colour.
- Affection of the external haemorrhoidal plexus which lies below the dentate line produces external haemorrhoids (Fig. 39.9). These are covered by skin.
- Interno-external haemorrhoids develop if both are present.

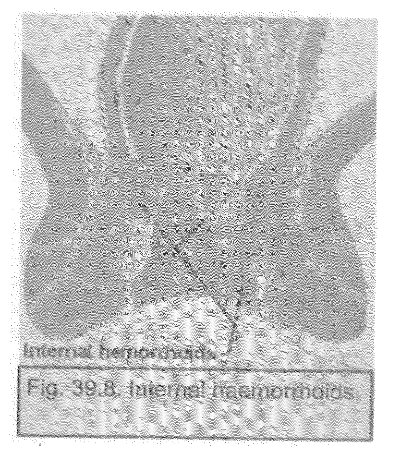


Fig. 39.8. Internal haemorrhoids.

Aetiology

Haemorrhoids may be classified into primary where there is no definite aetiological factor or secondary (much less frequent) where there is a definite causative factor.

In primary haemorrhoids. Predisposing factors are suggested:

1. Genetic factors. Haemorrhoids are more common in certain families, a fact which suggests that congenital weak mesenchyme may have a role.
2. Chronic straining at defecation.
3. Anatomical factors. Venous drainage of the anal cushions may contribute to the development of haemorrhoids as follows:
   - The anal canal cushions lie unsupported in the loose submucous layer. With aging and degeneration of the supporting elastic tissue, engorgement and dilatation result and haemorrhoids may develop.
   - The tributaries of the anal cushions penetrate the muscular wall of the rectum before joining the superior rectal veins, and so they are liable to be compressed during defecation especially if there is prolonged straining.

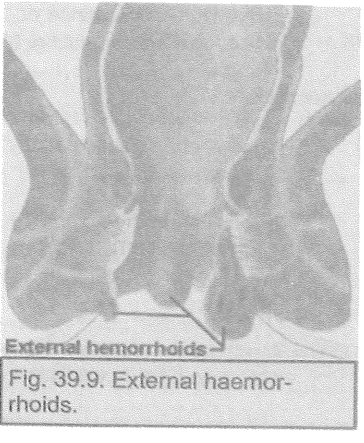


Fig. 39.9. External haemorrhoids.

- There are no valves in the portal circulation which leads to high hydrostatic pressure at the haemorrhoidal plexus.

Secondary haemorrhoids may occur with:

1. Pregnancy due to raised intra-abdominal pressure and the relaxing effect of progesterone.
2. Pelvic tumours particularly carcinoma of the rectum may obstruct tributaries of the superior rectal vein. Rectal carcinoma should be excluded in patients with bleeding per rectum, even if there are evident haemorrhoids.

Clinical features

Symptoms

1. Bleeding per rectum. This is the most common complaint. Haemorrhoidal bleeding is characterized by:
   - Occurs with straining, usually at the end of defecation.
   - Fresh bright red.
   - Jet or drops that are separate from stools.
2. Prolapse. The vascular cushions enlarge and start to descend below the dentate line. Piles are classified according to their degree of prolapse into:
   - First degree piles. There is no prolapse of piles. The patient may have no symptoms or may present by bleeding only. These piles are diagnosed only by proctoscopy.
   - Second degree piles. The piles prolapse only during defecation, but they are spontaneously reduced at the end of the act.
   - Third degree piles. There is prolapse of piles during defecation and the patient has to manually reduce them.
   - Fourth degree piles. There is permanent prolapse of piles.

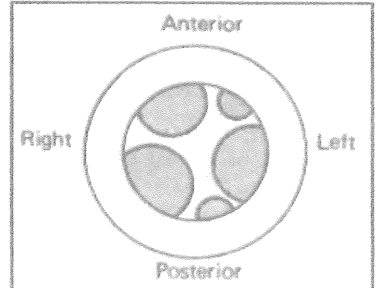


Fig. (39.10): Appearance of haemorrhoids at proctoscopy

3. Anal discharge and pruritus (itching). Patients who have prolapse of their piles beyond the anal verge, will complain of mucous discharge which may cause pruritus ani.
4. Pain and discomfort. Uncomplicated haemorrhoids are painless. The presence of pain indicates either a complication of the haemorrhoid or the presence of another problem, e.g., a fissure.

Examination

1. In early cases no abnormality can be seen outside the anal verge and only proctoscopy can reveal the presence of internal haemorrhoids (Fig. 39.10) which appear as bluish bulges below the anorectal ring.
2. In late cases prolapsing piles can be seen (Fig. 39.11) in one or more of the three primary positions. These are called mother piles. There may be daughter piles between the main 3 ones.
3. Uncomplicated piles are impalpable on digital rectal examination because they are compressible. This examination is, however, essential to exclude a palpable rectal cancer.

Fig. 39.11. Prolapsing haemorrhoids.

4. Sigmoidoscopy should be performed to exclude rectal cancer.

Complications

1. Profuse haemorrhage.
2. Anaemia.
3. Strangulation occurs when one or more of the internal haemorrhoids prolapse and become gripped by the external sphincter. This leads to interference of the venous return and so the haemorrhoids become very tense, swollen and very painful. It is said that strangulated haemorrhoids had cost Napoleon Waterloo battle and the French their empire.
4. Thrombosis. If strangulation is not relieved rapidly, thrombosis occurs and the affected haemorrhoid becomes dark purple or black (Fig. 39.12).
5. Fibrosis. Internal haemorrhoids sometimes become converted to fibrous tissue after thrombosis. A fibrous haemorrhoid is white in contrast to an adenoma which is bright red.
6. Ulceration may follow strangulation and thrombosis.
7. Gangrene. If the strangulation is very tight, the arterial supply of the haemorrhoid is constricted leading to a superficial slough or the whole haemorrhoid sloughs leaving an ulcer.
8. Suppuration is the result of infection of a thrombosed haemorrhoid.
9. Portal pyaemia is a rare complication of infected haemorrhoids.

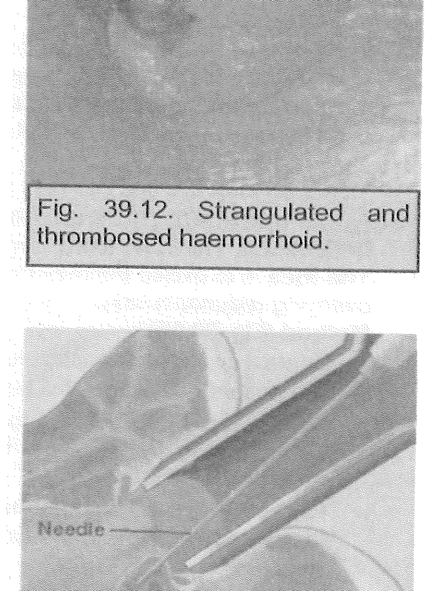


Fig. 39.12. Strangulated and thrombosed haemorrhoid.

Fig. 39.13. Injection sclerotherapy of haemorrhoids.

Treatment

Primary haemorrhoids

- For first and second degrees, conservative treatment, injection sclerotherapy, rubber band ligation, and photocoagulation are successful.
- For third and fourth degree haemorrhoids surgery is recommended.

For secondary haemorrhoids, treatment is directed to the cause.

Conservative treatment

Indications. Early cases.

Method

- High fibre diet.
- Small doses of laxatives.
- Avoidance of straining at defecation.
- Suppositories that contain decongestants and astringents.

Injection sclerotherapy

Indications. Bleeding first and second degree hemorrhoids.

The idea is to inject an irritant material in the submucosa at the pedicle of the haemorrhoids. The resulting fibrosis will obliterate the venous plexus and at the same time will pull the prolapsed cushions up.
Method (Fig. 39.13)

- This is an office procedure which does not need anaesthesia as the injection is performed in a non sensitive area.
- Three ml of 5% phenol in almond oil is injected at the upper end of each haemorrhoid in the submucosa (not in the vessels). The three haemorrhoids can be injected at the same time.
- The procedure can be repeated once or twice after 2 weeks.

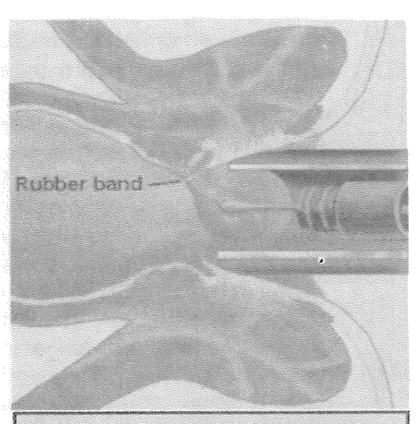


Fig. 39.14. Rubber band ligation of haemorrhoids.

Complications

- Severe pain if the injection is done at a low position in the sensitive anoderm.
- Necrosis of the overlying mucosa if the injection is superficial.
- If the injectito is deep it, may lead to haematuria.
- Allergic manifestations.
- Submucous abscess formation.

Rubber band ligation:

Indications: This procedure can be performed in second and early third degree haemorrhoids.

The idea is to place a tight elatic rubber band around the pedicle of the haemorrhoid leading to its ischaemic necrosis and later separation (Fig. 39.14).

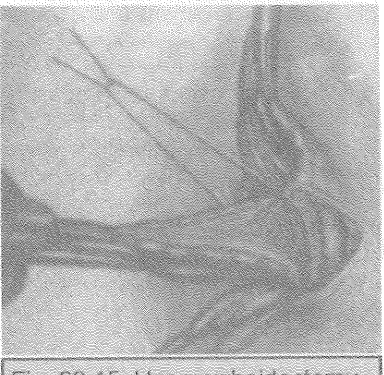
This is an office procedure which does not need anaesthesia.

Intra-red photocoagulation

Indications. The same as injection sclerotherapy.

The idea. Infrared photocoagulation can produce a tissue temperature of 100 °C which prodoues an area of coagulation necrosis. The dead tisues separate after 10-14 days leaving a granulation lined ulcer.

This is an effective and painless method.


Fig. 39.15. Haemorrhoidectomy.

Surgical treatment (haemorrhoidectomy)

Indication. Third and fourth degree haemorrhoids.

The idea is to excise the hypertrophoid vascular cushions with the overlying redundant skin.

Method. (Fig. 39.15)

- General or spinal anesthesia.
- The patient is palced in the lithotomy position.
- An Allis forceps is applied to the skin overlying each external haemorrhoids and an artery forceps on each internal haemorrhoids.

- Traction is applied on both the Allis and artery forceps, and a V shaped cut is made in the skin overlying the external haemorrhoid.
- Dissection exposes the lower end of the internal sphincter.
- The pedicle of the internal haemorrhoid is ligated by strong silk or transfixed by strong catgut.
- Each haemorrhoid is dealt with in the same manner and then excised 1 cm distal to the ligature.
- It is essential to leave islands of intact skin and mucosa between the individual pedicles.
- Postoperative strong analgesics.

Presentations of common anal conditions

Fissure
Pain with & after defecation.

Uncomplicated piles
Bleeding
Prolapse

Strangulated piles
Pain and prolapse

Abscess
Pain (persistent) and fever

Fistula
Discharge

Complications:

1. **Haemorrhage**. This may be reactionary or secondary. Reactionary haemorrhage is treated by giving morphia injection and applying local pressure by a piece of gauze. If the bleeding persists, surgical ligation of the bleeding point is mandatory. Secondary haemorrhage is more serious as there is secondary infection and the tissues are very friable. Conservative measures are tried at first, but if they fail, surgical ligation of the bleeding by underrunning sutures is performed. In some patients if sutures fail to stop the bleeding, a pack around a large rectal tube is inserted in the anal canal for 2-3 days.
2. **Retention of urine**. This is a common problem after many anal operations especially in elderly males. Conservative measures are tried at fist. An analgesic is prescribed for the pain, if there is an anal pack, it is removed, the patient is asked to get out of bed, a warm bath is advised, and an injection of a parasympathomimetic drug as carbachol is administered. If these measures fail, catheterization is advised.
3. **Anal stricture**. To avoid this complication intact areas of anal skin and mucosa should be left between the raw areas of excised haemorrhoids. Two weeks post-operatively a digital rectal examination is performed to check that there is no tendency
   to stenosis. If present, gradual anal dilation is started.
4. **Recurrence** is liable to occur in young patients and is usually due to enlargement of daughter piles. Injection treatment is advised.
5. One of the cutaneous wounds may not heal completely and leads to a **fissure**. Conservative treatment is usually successful. If it fails internal sphincterotomy solves the problem.

Treatment of prolapsed strangulated haemorrhoids:

- If the case is diagnosed early, surgical intervention under antibiotics is indicated.
- If the diagnosis is delayed, the tissues are friable and there may be secondary infection and, therefore, it is better to rely on conservative measures.
  - Rest in bed.
  - Antibiotics.
  - Analgesics.
  - Frequent warm baths.
  - Decongestive ointments and local compresses by lead subacetate lotion.

Acute perianal haematoma (thrombosed external haemorrhoid)

Pathogenesis

This is a very painful condition due to rupture of a dilated anal vein and its spread, secondary to straining at defecation, coughing or lifting a heavy weight. It leads to haemorrhage in the loose subcutaneous connective tissue usually in the lateral position.

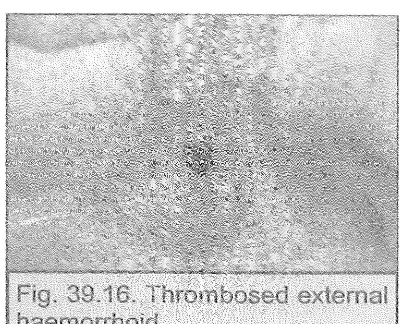


Fig. 39.16. Thrombosed external haemorrhoid.

Clinical features

It appears as a tense, tender, bluish swelling covered by smooth shining skin (Fig. 39.16).

Untreated, the haematoma usually resolves. It may, however, suppurate, ulcerate through skin with extrusion of the blood clot or fibrose and give rise to a cutaneous tag.

Treatment

If the patient presents early with severe pain the haematoma is evacuated.

Anorectal abscess

Aetiology

Primary anorectal abscess is due to infection of the anal glands or the skin glands.

The anal glands exist in a plane between the internal and external anal sphincters and communicate by their ducts to the anal mucosa at the level of the dentate line (Fig. 39.1). Infection of these glands by Gram-negative bacilli leads to the formation of an intersphincteric abscess which may spread (Fig. 39.17):

- Downwards → Perianal abscess
- Outwards → Ischiorectal abscess
- Inwards → Submucous abscess

Fig. 39.17. Origin of abscess and its spread.
1. Intersphincteric
2. Perianal
3. Ischiorectal
4. Submucous

In the majority of these abscesses, there is an inner opening in the anal canal and drainage of the abscess is usually followed by a fistula.

Infection of the apocrine glands or hair follicles of the perianal skin is the aetiology of perianal suppuration in 15-25% of cases. In such cases the causative organisms are usually Staphylococcal and there is no communication with the anal canal.

Secondary anorectal abscess

- Inflammatory bowel disease as in Crohn's disease and ulcerative colitis.
- Specific infections as tuberculosis.
- Anorectal carcinoma.
- Infection of a perianal haematoma, thrombosed haemorrhoids or anal fissure (fissure abscess).

Fig. 39.18. Types of anorectal abscesses.

Classification and clinical features (Fig. 39.18)

1. Perianal abscess (60%) is usually due to downward spread of an intersphincteric abscess or infection of a perianal haematoma. The abscess lies subcutaneously adjacent to the anal orifice. Pain and constitutional symptoms are not marked.
2. Ischiorectal abscess (30%) is usually due to lateral extension of an intersphincteric abscess. Fever is high with marked constitutional manifestations. The abscess presents as a large, indurated, tender swelling filling the ischiorectal space. There is throbbing pain and pitting oedema. If the abscess is not drained early, it will extend through the postsphincteric space to involve the other side leading to horse shoe abscess. The surgeon should not wait for fluctuation to drain this abscess.
3. Submucous abscess (5%). The abscess lies in the submucous space above the dentate line. The patient has severe pain and fever but nothing is seen outside the anal verge. This abscess is one of the causes of pyrexia of unknown origin. Digital rectal examination reveals a tender boggy swelling.
4. Pelvirectal abscess (5%). This is actually a pelvic abscess secondary to appendicitis, salpingitis or diverticulitis. The abscess is located between the upper surface of the levator ani and pelvic peritoneum. A rare cause of this abscess is due to injury of the levator ani muscle by an instrument during drainage of an ischiorectal abscess.

Treatment

- Urgent surgery (incision and drainage) under general anaesthesia is performed.
- The wound is packed and daily dressings are arranged.
- If the abscess is near the anal verge, the surgeon should look for an internal fistulous pening and if present, he should treat the patient as if having a fistula.
- The patient who had drainage of an anorectal abscess should be warned that he may develop an anal fistula.

Anal fistula

Definition

An anal fistula is a track lined by granulation tissue and extends from an opening at the skin of the perianal region to the cavity of the anal canal or rectum.

Aetiology

Most fistulae arise from a pyogenic intersphincteric abscess which has been allowed to develop to the point of spontaneous rupture or which has not been properly drained surgically.

Pathology

Factors that are blamed for persistence of anal fistulae

1. The anal glands act as a reservoir for infection.
2. The presence of an internal opening allows recurrent activation of infection with further build-up of pus which discharges both internally and externally.
3. Underlying specific diseases may be associated with persistent fistulae such as Crohn's disease, tuberculosis, and ulcerative colitis.
4. Faecal material may act as a foreign body but it is surprisingly rare to find such material on exploring the frstulous track.

Sometimes, there are multiple external openings but usually there is a single internal opening.

Classification

The outline of the fistulous track is considered in two planes: horizontal and vertical.

- The **horizontal extension** is governed by Goodsall's rule which states that the position of the internal opening in the circumference of the anal canal depends upon the relationship of the external opening to an imaginary line drawn between the ischial tuberosities bisecting the anus. Fistulas with external openings anterior to Goodsall's line have their internal openings on the same radius having short direct fistulous track, whereas fistulas with external openings behind this line communicate with an internal opening in the midline posteriorly Fig. 39.19).
- The **vertical axis** of the anal fistulous track is important to determine, before proper management to avoid injury of the sphincteric control of continence during surgery.
    - Standard classification divides anal fistulas into high and low according to the position of their internal opening in relation to the anorectal ring.
    - New classification. Anal fistulae are recently classified according to the course and relation of the fistulous track to the anorectal sphincter muscles. Almost all anal fistulae have their internal opening at the level of the dentate line communicating with an intersphincteric abscess cavity. According to the subsequent course of the fistulous track to the external opening, anal fistulae are classified into four types (Fig. 39.20):

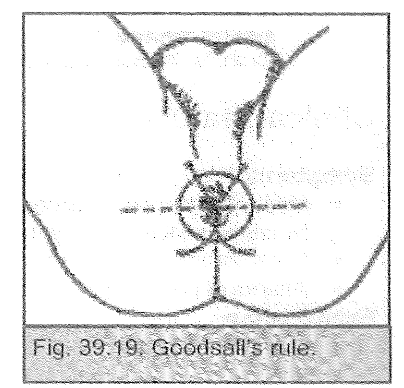


Fig. 39.19. Goodsall's rule.

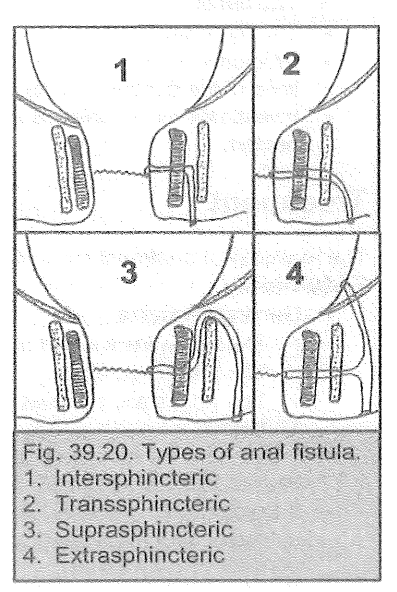


Fig. 39.20. Types of anal fistula.
1. Intersphincteric
2. Transsphincteric
3. Suprasphincteric
4. Extrasphincteric

- **Intersphincteric** fistula begins at the dentate line and extends to the perianal skin, traversing the plane between the internal and external anal sphincters.
- **Transsphincteric** fistula begins at the dentate line and traverses both the internal and the external anal sphincters, opening through the ischiorectal fossa in the perianal skin. The fistula may be complicated by additional blind tracks.
- **Suprasphincteric** fistula begins at the dentate line communicating with an intersphincteric abscess cavity. The track then traverses the entire thickness of the external sphincter and puborectalis muscle (hence supra-sphincteric). It further passes downwards across the ischiorectal fossa to open in the perianal skin.
- **Extrasphincteric** (supralevator) fistula usually traverses the ischiorectal fossa, through the levator ani muscle, connecting the rectal wall to the perianal skin having their track coursing outside the plane of the external anal sphincter. They usually have an intermediate communicating track passing through the external and internal sphincters to the original internal opening at the level of the dentate line of the anal canal. These fistulas are usually complex and have several causes, such as high anorectal abscess rupturing into the rectum, rectal trauma, rectal Crohn's disease or pelvic abscess.

Clinical features

Symptoms

- Most patients give a history of a previous perianal abscess which discharged spontaneously followed by intermittent or persistent discharge.
- Local soreness and pruritus ani.
- Attacks of perianal pain occur as recurrent abscesses build up.

Examination

- Single or multiple external openings next to the anal orifice (Fig. 39.21).
- If the fistula is active, granulation tissue may be seen at the opening and pus can be expressed. Alternatively the fistula may have healed temporarily.
- The perianal skin shows induration.
- On digital examination an internal opening can sometimes be felt.
- Proctoscopy may show the internal opening of the fistula usually at the bottom of an anal crypt at the level of the dentate line. Its relation to the anorectal ring must be clearly defined.
- Investigations by colonoscopy and/or barium enema are rarely needed if Crohn's disease is suspected.

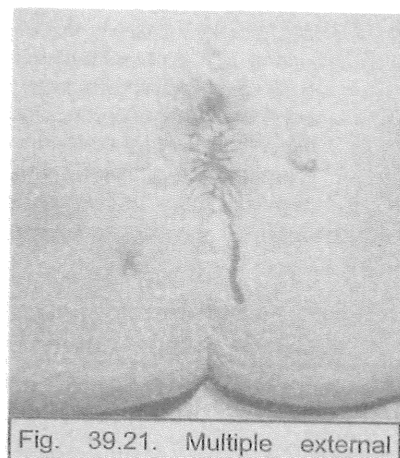


Fig. 39.21. Multiple external openings of an anal fistula. They have one common internal opening in midline posteriorly at the level of dentate line.

Treatment

The mainstay of treatment for anal fistula is eradication of sepsis with preservation of anorectal function.

Fistulotomy

- The fistula track is laid open, the track is curetted, and the edges are trimmed so as to leave an open well-drained wound that heals by secondary intention.
- Fistulotomy for an intersphincteric fistula entails division of part of the internal sphincter for deroofing the fistulous track. There is no disturbance of continence following this procedure.
- Fistulotomy for transphincteric fistula entails deroofing the track by division of part of the internal sphincter and the superficial part of the external sphincter. Some minor temporary disturbance of continence may follow.
- Fistulotomy for suprasphincteric fistula will include division of the entire sphincter anal mechanism and would surely produce incontinence. A staged fistulotomy is performed instead of single-stage procedure. The initial stage is to deroof the lower part of the intersphincteric component of the fistubus track by dividing the internal sphincter up to the internal opening at the dentate line. A seton suture is left in place through the rest of the track, snugly tied around the puborectalis and external sphincter. Two weeks later the rest of the track is laid open guided by the seton suture. The fibrosed adherent ends of the sphincter are claimed to be prevented from retraction widely apart maintaining the mechanism of continence.
- Extrasphincteric fistulae are often complicated and complex. Treatment is usually tailored to the undenying cause, but usually includes proximal colostomy as well as staged fistulotomy.

Fistulectomy is complete excision of the fistulous track.

Faecal incontinence

Faecal incontinence is defined as the inability to retain the rectal contents at will.

Causes

1. Damage to the anal sphincter mechanism
   - Obstetrical trauma causing complete perineal tear.
   - Surgical trauma, e.g., during surgery for a high anal fistula.
   - Accidental trauma.
2. Complete rectal prolapse. The prolapsing rectum stretches the anal sphincters damaging them. Excessive descent of the perineum with straining is a common accompanying feature, which may produce pudendal nerve traction neuropathy.
3. Idiopathic faecal incontinence.
4. Neurological diseases
   - Trauma or tumours affecting the second, third and fourth sacral nerves.
   - Diabetic autonomic neuropathy.

   Diarrhoea accentuates incontinence, and may even produce it in patients with borderline weak sphincters.

Assessment

1. Clinical assessment for:
   - Degree of incontinence, e.g., incontinence to flatus only, to flatus and fluid stools, or to solid stools as well.
   - History of trauma, surgery or difficult labour.
   - Rectal prolapse.
   - Sphincter contraction.
   - Neurological assessment.
2. Manometry to detect rectal function, length of the anal sphincters, and their strength.
3. Electromyography assesses the function of the pudenal nerve.
4. Defaecography to assess the anorectal angle.
5. Trans-anal ultrasound and MRI can accurately localize the site of sphincteric damage.

Treatment

Conservative treatment is indicated in mild cases.

- Constipating agents and dietary manipulations to thioken the stools.
- Anal sphincters and pelvic floor exercises.
- Evacuating the bowel completely with a glycerine suppository in the morning so as to avoid soiling later in the day.

Surgery

- Repair of divided sphincters.
- Rectopexy will restore continence to 50% of those having complete rectal prolapse.
- Postanal repair is indicated for idiopathic incontinence. The aim of the operation is to restore the right angle of the anorectal junction. The levator ani and sphincters are plicated behind the anorectal junction, pushing it forwards.

Malignant tumours

Pathology

The commonest type is squamous cell carcinoma arising from the anoderm. Less commonly adenocarcinoma arises above the dentate line.

Metastases may develop in the inguinal lymph nodes if the tumour is below the dentate line or in the paraaortic lymph nodes if the tumour is above the dentate line.

Clinical features

- The patient presents by a lump, pain or discharge.
- Digital examination usually reveals an indurated ulcer that bleeds easily
- Inguinal lymph nodes are examined for metastases.

Biopsy is essential before institution of treatment.

Treatment

Carcinoma of the anal verge

Wide local excision with 2.5 cm safety margin. If there are metastatic lymph nodes inguinal block dissection is performed.

Carcinoma of the anal canal

One of two lines may be performed.

- Chemoradiation. A course of combination chemotherapy is followed by radiotherapy (squamous cell carcinoma is radiosensitive). The patient is examined after 4-6 weeks. If there is any evidence of residual tumour, abdominoperineal resection is performed.
- Abdominoperineal resection of the anal canal and rectum with a terminal colostomy is indicated for large tumours.

REVIEW SUBJECTS

CHAPTER CONTENTS

All the individual items in this chapter have been discussed in detail elsewhere in this book. The aim is to give a collective review of some important surgical problems. Stress will be made on their main features.

Abdominal Injuries

Mechanism of Injury

1. **Blunt injuries** are due to direct blows to the abdomen or more commonly to motor car accidents due to sudden deceleration which causes stress to areas of junction between freely mobile intraperitoneal organs and those with a fixed retroperitoneal position. Common sites of injury include the renal pedicle, the duodenojejunal flexure, the ileocaecal area or the neck of the pancreas. Sudden compression of the abdomen may also cause laceration of the liver or spleen.
2. **Penetrating injuries** are due to stabs or gunshots. The severity of injury with gunshots is determined by the formula: **[Kinetic energy = MV^2/g]**. Where m is the mass of the bullet, v is the velocity and g is the acceleration of gravity. When a bullet strikes soft tissues, shock waves spread out of the missile tract causing extensive damage to the surrounding tissue and even to areas far from the primary tract (see chapter 1).
3. **Blast injuries**. The blast waves cause shear waves which may lead to submucosal haemorrhages, mesenteric tears or perforation near the ileocaecal area. Solid organs as the liver may be severely lacerated. In addition to the blast wave, the produced missiles after the explosion can cause different blunt or penetrating abdominal injuries.

Possible intraabdominial injuries

- Injury to solid organs e.g. the liver, the spleen or the mesentery leading to internal haemorrhage.
- Injury to hollow organs e.g. the stomach, duodenum, the small bowel or the colon leading to peritonitis.
- Retroperitoneal injuries e.g. the pancreas, the kidneys or the major blood vessels.

Important points

- The site of injury and the localization of the physical signs may give some clue to the diagnosis e.g. the presence of hypovolaemic shock with tenderness and rigidity in the right hypochondrium usually denote a liver injury.
- Trauma to the lower thoracic cage may lead to splenic or hepatic injuries.
- In bowel injuries the clinical picture may not be apparent on initial examination.
- Stab wounds to the abdomen are non penetrating in one third of cases, penetrating but not causing any intraabdominal injury in one third, and will cause an intraabdominal injury in one third of patients.

Investigations

Laboratory investigations

- Blood picture and haematocrit value. A declining haemoglobin and haematocrit denote bleeding.
- A rising leucocytic count points to peritonitis.
- High serum amylase level suggests pancreatic injury.

Radiological

- Plain chest and abdominal X-rays (only in haemodynamically stable patient).
  - This may reveal fracture of the lower ribs or pelvis or the presence of a foreign body.
  - Free air under the right cupola of the diaphgram denotes injury of a hollow organ.

Abdominal ultrasound; Focused abdominal sonography of trauma (FAST)

- This investigation is non-invaisve, quick, inexpensive and can be performed at the bedsite.
- It has a sensitivity of 85-95% for the detection of intraabdominal fluid or blood e.g. perisplenic or perihepatic haematoma.
- The disadvantages are that it is operator dependent and it is not sensitivie for the diagnosis of bowel perforation.

CT abdominal scan

- This should be only performed in a stable patient as it entails the transfer of the patient to the radiology department and it takes sometime to be performed.
- It is very accurate in detecting injury to solid organs and in the grading and follow-up of these injuries.
- It is also sensitive in the diagnosis of retroperitoneal and diaphragmatic injuries.
- It is not sensitive in the detection of bowel injuries or acute pancreatic injuries.

Diagnostic peritoneal lavage (DPL)

Indications

Blunt abdominal trauma in an adult, associated with:

1. Suspicion of organ injury with equivocal signs.
2. Unreliable abdominal examination because the patient is unconscious, e.g., head trauma, or drug or alcohol intoxication.
3. Unexplained hypotension that may be caused by blood loss.

Contraindications

1. Evident intra-abdominal organ injury that requires laparotomy.
2. Pregnancy.
3. Liver cirrhosis.
4. Severe obesity.
5. Prior abdominal surgery.

Procedure

1. Abdomen is prepared with an antiseptic solution and is draped with sterile towels.
2. Local infiltration of local anesthetic, e.g. lidocaine in the midline below the umbilicus.
3. 2-3 cm skin incision followed by a 1 cm incision in the linea alba.
4. Peritoneum is entered with a dialysis catheter.
5. The tube is directed posteriorly and inferiorly into the pelvis.

6. Aspiration with a syringe. Gross blood, or gross enteric contents are indications for immediate laparotomy.
7. If neither blood nor enteric content is aspirated, I L of warm saline is instilled into the peritoneum by intravenous tubing.
8. After waiting for 5 minutes the empty saline bottle is placed down in a dependent position to siphon the lavage fluid out of the abdomen.
9. A sample of the fluid is sent to the laboratory. Positive findings that diagnose an intra-abdominal surgery, and thus require laparotomy are:
    a. Red blood cell count >1000000/ml.
    b. White blood cell count> 500/ml.
    c. Elevated amylase.
10. The catheter is removed, and the linea alba and skin are closed with sutures.

Diagnostic laparoscopy.

Treatment of abdominal injury

- Follow the general measures for trauma patients (Chapter 2).
- The surgeon should decide whether the patient needs an urgent Laparotomy or the patient's condition is stable so that he can do the necessary investigations and follow the patient's condition.

Urgent laparotomy without losing valuable time doing unnecessary investigations is indicated for:

1. General and local clinical manifestations of intra-abdominal bleeding, e.g., pallor, tachycardia, hypotension, together with abdominal tenderness, rigidity and distension.
2. General and local clinical manifestations of peritonitis.
3. Stab wounds with a protruding viscus. This means that injury extended deeper than the parietal peritoneum.
4. All missile injuries of the abdomen.

N.B.: There is now a growing tendency to treat many patients with hepatic and splenic injuries conservatively.

Acute abdomen

The items of this chapter are discussed in details in their corresponding chapters. The intention is to discuss in brief the important points in clinical presentation and investigations.

Definition Acute abdomen is a term applied to acute abdominal pain.

Clinical features

In the majority of patients, a proper diagnosis is reached by means of a careful history and complete physical examination supplemented by a few simple laboratory tests.

Personal history. The age, sex, marital status, residence and menstrual history are recorded.

Present History

Symptoms

1. **Acute pain** is usually the most important symptom. It is to be noted that there are two types of abdominal pain, viz, visceral or somatic. Visceral pain is usually due to

distension of a hollow viscus. It is felt in the segment of the abdominal wall having the same nerve supply of the affected organ and it is vague ill defined pain, and so;

a. Foregut pain (stomach and duodenum) is felt in the epigastrum.
b. Mid-gut pain (jejunum to transverase colon) is felt in the peri-umbilical region.
c. Hind-gut pain (transverse colon to anal canal) is felt in the lower abdomen.

Somatic pain is due to irritation of the parietal peritoneum and so it is well localized over the affected organ. The presence of these two types of pain explains why the initial pain of acute appendicitis is vague and is felt in the umbilical region, but later it becomes localized to the right iliac fossa.

- Abdominal pain may be referred to other regions, e.g., renal ache or ureteric colic may be referred to the groin or testis along the distribution of the genitofemoral nerve, and biliary colic may be referred to the lower angle of the right scapula (seventh to ninth thoracic nerves).
- Sudden onset of pain denotes haemorrhage, perforation or torsion while gradual onset is due to inflammation.

2. **Vomiting** usually occurs once or twice after the onset of pain. Frequent and profuse vomiting is serious. Forcible vomiting is typically seen in intestinal obstruction and is due to contraction of the stomach and abdominal muscles. Regurgitant vomiting is merely passive return of gastric contents without any force and it occurs in acute gastric dilatation, paralytic ileus or peritonitis.
3. **Bowels**: Constipation is the rule in most cases of acute abdomen. Tenesmus may be present in pelvic appendicitis. Mesenteric thrombosis may be accompanied by bleeding per rectum.
4. **Urinary problems** and menstrual disturbances should be inquired about.

Signs

1. Full general examination should be performed checking the vital signs, respiratory rate and looking for pallor, jaundice and dehydration.
2. Meticulous abdominal examination by inspection, palpation, percussion and auscultation is performed. Do not forget to examine the spine, the scrotum, the hernial orifices and to do per rectal examination.

Classification of causes

According to the mode of presentation.

1. Colics. Intestinal, appendicular and biliary.
2. Inflammations. Appendicitis, cholecystitis, pancreatitis, diverticulitis, and Meckel's diverticulitis.
3. Perforations. Perforated appendix, peptic ulcer, gall bladdeh diverticulum, and typhoid ulcer of the small bowel.
4. Intestinal obstruction. Simple, strangulation, and paralytic ileus.
5. Internal haemorrhage.
6. Urological causes. Calculi and inflammations.
7. Gynaecological causes
   - Mid menstrual pain.
   - Twisted ovarian cyst.
   - Dysmenorrhoea.
   - Ruptured ectopic pregnancy.
   - Ruptured ovarian cyst.
   - Pelvic inflammatory disease.
8. Medical causes of acute abdomen:

- Severe gastroduodenitis.
- Mediterranean fever.
- Activity of a peptic ulcer.
- Intermittent porphyria.
- Enterocolitis.
- Basal pneumonia and pleurisy.
- Uraemia.
- Acute myocardial infarction.
- Diabetic ketoacidosis.
- Alcoholic hepatitis.

According to site

- **Upper abdominal**
    - Perforated peptic ulcer.
    - Leaking abdominal aortic aneurysm.
    - Biliary colic and acute cholecystitis.
    - Mesenteric vascular occlusion.
    - Acute pancreatitis.
    - Acute myocardial infarction.
- **Mid-abdominal**
    - Mesenteric vascular occlusion
    - Intestinal obstruction
- **Right lower abdomen**
    - Acute appendicitis.
    - Mesenteric adenitis
    - Regional ileitis (Crohn's disease)
    - Right ureteric colic
    - Colitis
    - Meckel's diverticulitis
- **Left lower abdomen**
    - Colonic diverticulitis
    - Left ureteric colic
    - Colitis
- **Pelvic**
    - Mid menstrual pain
    - Pelvic inflammatory disease
    - Proctitis
    - Cystitis
    - Complicated ovarian cyst
    - Prostatitis
    - Ectopic pregnancy
    - Pelvic appendicitis
- **Abdominal and back pain**
    - Biliary colic and acute cholecystitis
    - Acute pancreatitis
    - Renal and ureteric colic
    - Leaking abdominal aortic aneurysm
    - Posterior duodenal ulcer penetrating pancreas.
    - Spine diseases with radicular pain that radiates forwards.

Some common causes of acute abdomen

Acute appendicitis

- It can affect any age, but is commonest in the second and third decades.
- There is usually a characteristic shifting pattern of pain from the centre to the right lower abdomen.
- Pain always occurs before vomiting.
- Diarrhoea is usually against the diagnosis.
- Anorexia and nausea are almost always present.
- The clinical picture may be variable (retrocaecal, pelvic, pregnancy and children).
- Investigations are done in difficult cases:
    - Leucocytic count: Polymorphnuclear leucocytosis and shift to the left are usually present. Their absence, however, is not a clue against appendicitis.
    - If a ureteric calculus is suspected, urine examination, plain X-ray UT, IVU and ultrasound are diagnostic.
    - In females, if a tubo-ovarian or uterine problem is suspected, a pelvic ultrasound and laparoscopy are helpful.

Acute cholecystitis

- The initial pain is diffuse and colicky in the upper abdomen. Later it localizes in the right hypochondrium.
- It is difficult to palpate the gall bladder (20%), due to the overlying tenderness and rigidity.
- Ultrasound is diagnostic:
    - Gall stones are detected in 95% of cases.
    - Distension of the gall bladder.
    - Thickened walls.
    - Pericholecystic fluid collection.
    - Localized tenderness.
- HIDA scan is very helpful. In acute cholecystitis, the gall bladder is not visualized as the isotope fails to enter it because the cystic duct is blocked.

Acute pancreatitis

- Severe epigastric pain that increases in intensity.
- Pain is referred to the back.
- Profuse vomiting is a prominent feature. When the stomach becomes empty, the patient starts retching.
- The patient may be shocked.
- Tenderness and guarding are slight.
- Serum amylase rises within 12 hours and returns to normal within 2-3 days. It has no predictive value. Many other conditions raise the serum amylase.
- Plain X-ray of the abdomen may exclude other problems.
- CT may reveal enlargement of the pancreas, peripancreatic fluid collection, or pancreatic necrosis.

Acute diverticulitis

- Rare before the age of 40.
- Sigmoid colon is the commonest site.
- Diagnosis relies mainly on the clinical picture.
- Gastrografin enema may be helpful.
- Barium enema should be postponed till after resolution of the acute attack.

- CT may reveal localized thickening of the colonic wall, density in the pericolic fat or a pericolic abscess.

Perforated peptic ulcer

- May be related to the intake of non-steroidal anti-inflammatory drugs (NSAIDs) or corticosteroids.
- The onset is sudden,
- Perforated DU:GU = 6:1.
- The ulcer may be acute or chronic.
- Board like rigidity is present.
- In the elderly, the symptoms and signs are not marked.
- Plain X-ray of the chest in the erect position reveals free gas under the cupola of the diagram in 60- 85% of cases.
- If plain X-ray does not show free gas, gastrografin meal will reveal leakage of the contrast.
- Differential diagnosis
    - Causes of acute upper abdominal pain that were mentioned earlier.
    - Acute appendicitis. In some cases the leaking fluid from a perforated ulcer trickles along the right paracolic gutter to the right iliac fossa to simulate appendicitis.

Perforated typhoid ulcer

- Perforation usually occurs during the 3rd or 4th week of fever.
- Fever, malaise and headache are present.
- Severe local pain and tenderness.
- Collapse is usually present.

Simple bowel obstruction

- Colicky abdominal pains
- Vomiting
- Abdominal distension
- Absolute constipation.
- Plain X-ray will reveal distended loops or fluid levels.
- Small bowel enema (enteroclysis) in cases of doubtful small bowel obstruction may be indicated.
- Barium enema in large bowel obstruction is diagnostic.

Strangulation intestinal obstruction

- Severe pain which is persistent and is not relieved by nasogastric suction.
- Localized tenderness and guarding.
- Fever and tachycardia.
- Leucocytosis.

Mesenteric ischaemia

- This is actually a type of strangulation intestinal obstruction.
- Suspect the diagnosis in patients over 50 years with valvular or atherosclerotic heart disease, arrhythmias, hypotension, hypovolaemia, myocardial infarction, or polycythaemia.
- The pain is out of proportion to abdominal findings.
- Haematemesis and melaena may be present.
- Plain X-ray may reveal ground glass appearance.

- Ultrasound and CT scans may show the occluding thrombus, bowel wall oedema or abnormal gas patterns.
- Aortogram. A sure diagnosis is obtained by a retrograde transfemoral aortogram with the catheter tip lust above the origin of the superior mesenteric artery. A lateral view can usually differentiate between embolism and thrombosis. If proximal arterial occlusion is not seen, a selective arteriogram reveals distal emboli, distal thrombi, small vessel disease, or venous occlusion.
- If the clinical picture is suggestive of mesenteric ischaemia and there are no facilities for these sophisticated investigations, laparotomy is performed without delay.

Pseudo-obstruction

This term includes a group of disorders which give the clinical picture of intestinal obstruction, but without a demonstrable organic lesion. Pseudo-obstruction is caused by the following disorders:

- Elderly patients with cerebrovascular accidents.
- Endocrine disorders, e.g., myxoedema.
- Neurologic disorder, e.g., Parkinsonism.
- Drugs, e.g., atropine like and tranquilizers.
- Electrolyte disturbances, e.g., hypokalaemia.
- Metabolic disturbances as in uraemia and diabetic ketoacidosis.
- Shock, e.g., from burns and septicaemia.
- Retroperitoneal irritation by collection of blood or urine, or by acute pancreatitis.

Ruptured ovarian cyst

- There is lower abdominal pain, tenderness and guarding.
- No toxaemia.
- Abdominal and pelvic ultrasound examinations are diagnostic.

Torsion of an ovarian cyst

- Severe lower lateral abdominal pain.
- Adnexal mass may be palpable.
- Ultrasound is diagnostic.
- Laparoscopy is diagnostic and therapeutic.

Pelvic inflammatory disease (PID)

- Suspect the disease in females in the reproductive period.
- UD increases the possibility.
- Inflammation is usually bilateral, but may be severer on one side.
- Streptococci, E. coli and anaerobes are the usual causative organisms.
- There is lower abdominal tenderness and guarding with high pyrexia.
- Peritoneal signs in the upper abdomen suggest leakage or rupture.
- Per vaginal examination and movement of the cervix are tender.
- High vaginal swab for culture and sensitivity.
- Ultrasound.

Ruptured ectopic pregnancy

- Risk factors for ectopic pregnancy include prior salpingitis, tubal ligation, tubal repair, IUD and prior ectopic pregnancy.
- History of menstrual abnormalities may be present.
- Severe lower abdominal pain.
- Pallor is usually a striking feature.

- Abdominal examination reveals tenderness and guarding.
- Per vaginal examination reveals tender cervix.
- Chorionic gonadotropin testing is positive.
- Ultrasound. The presence of a gestational sac in the uterus is against the diagnosis. Free blood is present in the pelvis.
- Laparoscopy is very helpful.

Common acute abdominal conditions in infants and children

- Acute appendidtis:
    1. The diagnosis is difficult, and early perforation is common.
    2. Diarrhoea may be present.
- Non-specific mesenteric lymphadenitis: These patients are usually diagnosed as acute appendicitis.
- Intussusception: This presents by colicky abdominal pains, constipation and bleeding per rectum.
- Intestinal volvolus.
- Meckel's diverticulitis,
- Primary peritonitis.

Investigations of acute abdomen:

Enthusiasm for modern investigations should not overwhelm good clinical judgement. In the majority of cases accurate clinical examination supplemented by few tests can confirm the diagnosis. The following are some useful investigations.

Laboratory investigations

1. Full blood picture.
2. Urea and electrolytes.
3. Blood sugar.
4. Serum amylase.
5. Urine analysis.

Radiological investigations

1. Plain X-ray of the chest in the erect position may show free gas under the cupola of the diagram in perforation of a viscus. A basal pneumonia will be detected.
2. Plain X-ray of the abdomen may reveal:
    - Calculi of the urinary tract.
    - Distended loops of bowel or fluid levels in intestinal obstruction.
3. Abdominal ultrasound can diagnose the following:
    - Acute calcular cholecystitis. Stones are detected in 95% of cases. The gall bladder is distended, its wall is thickened and subserosal oedema may be visualized.
    - Acute pancreatitis. Enlargement of the pancreas, pancreatic pseudocyst or abscess and pancreatic necrosis can be detected.
    - Leaking aortic aneurysm.
    - Distended pelvicalyceal system in cases where ureteric stones cause colic.
    - Gynaecological disorders. Ruptured ovarian cyst, twisted ovarian cyst, ectopic pregnancy and pelvic inflammatory disease are easily detected by pelvic ultrasound.
4. **CT scan of the abdomen:** The main advantage of CT scan is that its picture is neither affected by obesity nor by the presence of ileus. It is very helpful for the diagnosis of the following conditions:

- Acute pancreatitis. CT reveals the extent of enlargement of the pancreas. It visualizes the presence of gas, fluid or blood around the pancreas. CT with contrast can detect pancreatic necrosis.
- Retroperitoneal haemorrhage.
- Bowel infarction. CT may show gas which may be intramural, in the mesenteric veins or in the portal vein. The bowel wall is thickened. Furthermore, the mesenteric thrombus may be visualized.
- Splenic infarction.
- Diverticulitis. Inflammation of the bowel wall with thickness is detected. An abscess can be visualized.
- Localized fluid collection or free fluid in the peritoneal cavity.

Diagnostic laparoscopy:

Diagnostic laparoscopy is valuable especially for gynaecological problems.

Upper gastrointestinal haemorrhage (haematemesis and melaena)

Definitions

Upper GI haemorrhage is usually due to lesions above the ligament of Treitz (end of duodenum). It manifests by haematemesis and/or melaena.

1. **Haematemesis** means vomiting of blood which may be of a coffee ground material if it is small in amount or bright red blood if it is of a large volume.
2. Melaena is passage of black tarry stools. In most cases it is caused by bleeding from the upper SI tract. Rarely, however, melaena arises below the ligament of Treitz, e.g., from lesions of the small bowel as neoplasms, bleeding Meckel's diverticulum, intussusception, Crohn's disease, mesenteric vascular occlusion and bleeding typhoid ulcers.
3. Occassionally upper GI bleeding is so massive and the SI transit is so fast to the extent that it manifests by fresh bleeding per rectum.
4. Minor trickling of blood from any part of the gut does neither manifests in vomitus nor in change of colour of stools. This is called 'occult SI bleeding" and will be discussed later in this chapter.

Causes

Oesophageal causes

- Oesopphageal varices.
- Reflex oesophagitis.
- Mallory Weiss syndrome. A longitudinal mucosal tear occurs in lower oesophagus due to severe inco-ordinated vomiting.

Gastric causes

- Multiple gastric erosions.
- Acute haemorrhagic gastritis.
- Chronic gastric ulcer.
- Begin or malignant neoplasms of the stomach.

Duodenal causes

- Acute duodenal ulcers
- Chronic duodenal ulcers

General causes

Blood diseases as purpura and haemophilia.

According to the data of the Casualty Department of Kas El-Aini Hospital, the commonest four causes of upper GI haemorrhge are in the following order:

1. Oesophageal varices.
2. Acute gastric erosions usually caused by administration of NSAIDs.
3. Acute haemorrhagic gastritis,
4. Chronic duodenal ulcer.

Management of acute upper GI bleeding

In emergency situations like in cases of severe GI bleeding treatment should not await a diagnosis. Life saving resuscitative measures should be initiated immediately and are then followed by diagnosis and definitive treatment.

1. Estimation of severity of bleeding and resuscitation
2. Localization of the site and cause of bleeding.
3. Treatment of specific lesions.

Estimation of severity of bleeding and resuscitation

- Admit to hospital. Severe bleeding cases require CU management.
- Initial then repeated clinical and haematocrit assessment.
- Insert two peripheral venous lines and withdraw blood for cross-matching and blood tests.
- Insert a Foley catheter. Urine output is the best monitor of tissue perfusion.
- A central venous line is needed for monitohng of severe cases.
- IV sodium containing fluids, e.g., Ringer's lactate are started until blood is available.
- A naso-gastric tube is inserted for all cases.
- Correct coagulopathy by fresh frozen plasma and by giving the missing factors, e.g., cryoprecipitate (factor VIII) for haemophilia, platelet concentrate for purpura, or IV vitamin K for hypoprothrombinaemia.
- A team approach that includes a gastroenterologist, a surgeon with expertise in GIT surgery, and skilled nurses gives better results.
- A major cause of morbidity and mortality is aspiration of blood. To prevent this complication in patients with altered mental status, endotracheal intubation should be considered.

Localization of the site and cause of bleeding

History

- Previous attacks and their management.
- Hepatitis and Shistosomiasis.
- Medications, particularly NSAIDs.
- Peptic ulcer symptoms.
- Bleeding tendency.

Examination

- Stigmata of cirrhosis; spider angiomata, jaundice, gynecomastia, palmar erythema, testicular atropy, splenomegaly, ascites, cirrhotic liver.
- Surgical scars.
- Tenderness.

Laboratory tests

- Haemoglobin percent and haematocrit value will show evidence of haemodilution after 3 hours.

- Liver function tests will be disturbed in patients with cirrhosis and oesophageal varices.
- Blood urea and creatinine.
- Exclude causes of generalized bleeding tendency by the coagulation tests.

Fibreoptic Endoscopy

Endoscopy is the most important test. It should be performed as early as possible once the patient has been resuscitated. The procedure is done under a mild sedative as diazepam.

In the majority of cases (90-95%) endoscopy will establish the source of bleeding and it may re vea the actual bleeding spot. Moreover, in cases of double lesions, endoscopy will tell which one is bleeding.

Endoscopy can also be used therapeutically to stop the bleeding.

Barium radiography is loosing favour because it is less accurate than endoscopy.

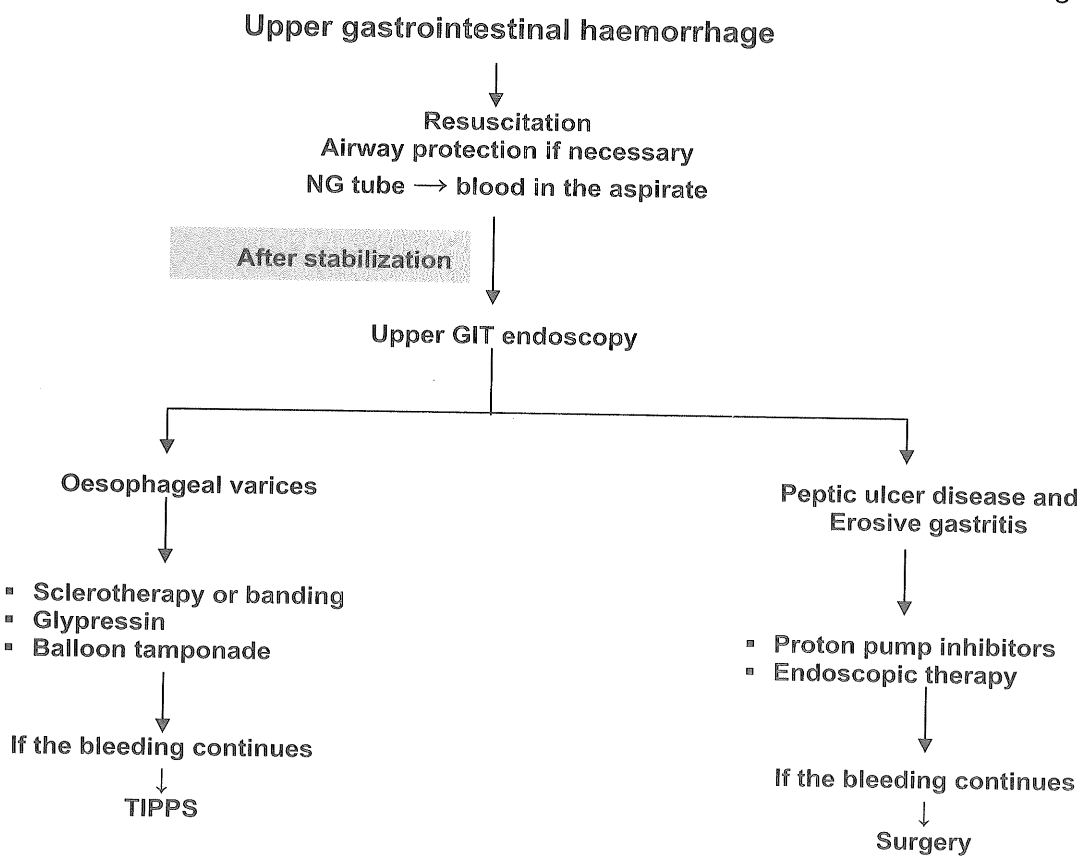
Angiography

In difficult situations where radiology or endoscopy fails to diagnose the lesion that causes bleeding, it may be necessary to resort to coeliac angiography to pinpoint the source of the bleeding, e.g. angiomatous malformation of the stomach. Angiography needs to be performed during active bleeding.

Treatment of specific lesions

- Bleeding oesophageal varices (Chapter 31)
- Bleeding peptic ulcers, erosive gastritis and multiple gastric erosions (Chapter 30).

Fig. (40.1) is an algorithm for the management of upper gastrointestinal haemorrhage.



Bleeding per rectum

Haematochezia is fresh bleeding per rectum.

Causes

1. The commonest causes are lesions of the anal canal as haemorrhoids and anal fissure.
2. Generalized causes as thrombocytopenia or leukaemia.
3. Colorectal causes:
   - Congenital lesions. Familial polyposis coli.
   - Inflammatory lesions
     - Acute bacillary dysentery.
     - Amoebic dysentery.
     - Bilharzial colitis.
     - Ulcerative colitis.
   - Vascular lesions as ischaemic colitis.
   - Diverticular disease of the colon.
   - Neoplastic lesions. Benign and malignant neoplasms of the colon and rectum.
   - Angiomatous malformation of the colon.

Hemorrhoidal bleeding is the commonest cause

Massive bleeding per rectum in adults

- Diverticula.
- Ulcerative colitis.
- Ischemic colitis.
- Angiodysplasia.
- Massive bleeding from upper GIT.

Massive bleeding per rectum in children

Meckel's diverticulum

Facts about bleeding per rectum

- Spontaneous remission rate is 80%. Bleeding has usually ceased by the time the patient presents to hospital.
- No source of bleeding can be identified in 12% of cases.
- Bleeding is recurrent in 25% of cases.
- Lower GIT bleeding is more difficult to diagnose than upper SIT bleeding.

Management of bleeding per rectum

- Minor bleeding is treated on elective basis. There is enough time for meticulous examination and investigations to reach a diagnosis before starting treatment.
- Massive bleeding per rectum is treated on an emergency basis as mentioned earlier for upper SI bleeding.

Clinical features

- Haemorrhoidal bleeding is characterized by:
  - Fresh bright red.
  - Jet or drops separate from stools.
  - Occurs with straining usually by the end of defecation.
- Patients with familial polyposis usually present in the second decade by loose stools, attacks of lower abdominal pain, diarrhoea and the passage of blood and mucous.

- In acute dysentery the main symptoms are abdominal pain, tenesmus and bloody diarrhoea.
- In ulcerative colitis there is a long history of diarrhoea with rectal discharge of mucous or blood.
- Patients with ischaemic colitis are usually elderly and complain of left sided abdominal pain and bright red rectal bleeding.
- In carcinoma of the colon or rectum the patient may complain of a recent change in the bowel habits, attacks of large bowel obstruction or bleeding per rectum. It should be stressed that the mere presence of haemorrhoids in an elderly patient does not ascertain that they are the cause of bleeding. Haemorrhoids and carcinoma of the rectum can coexist and it is a grave mistake to treat such a patient with haemorrhoidectomy leaving the carcinoma to advance to inoperability.
- In divertjcular disease and vascular malformations of the colon, clinical examination is usually irrelevant.

Investigations

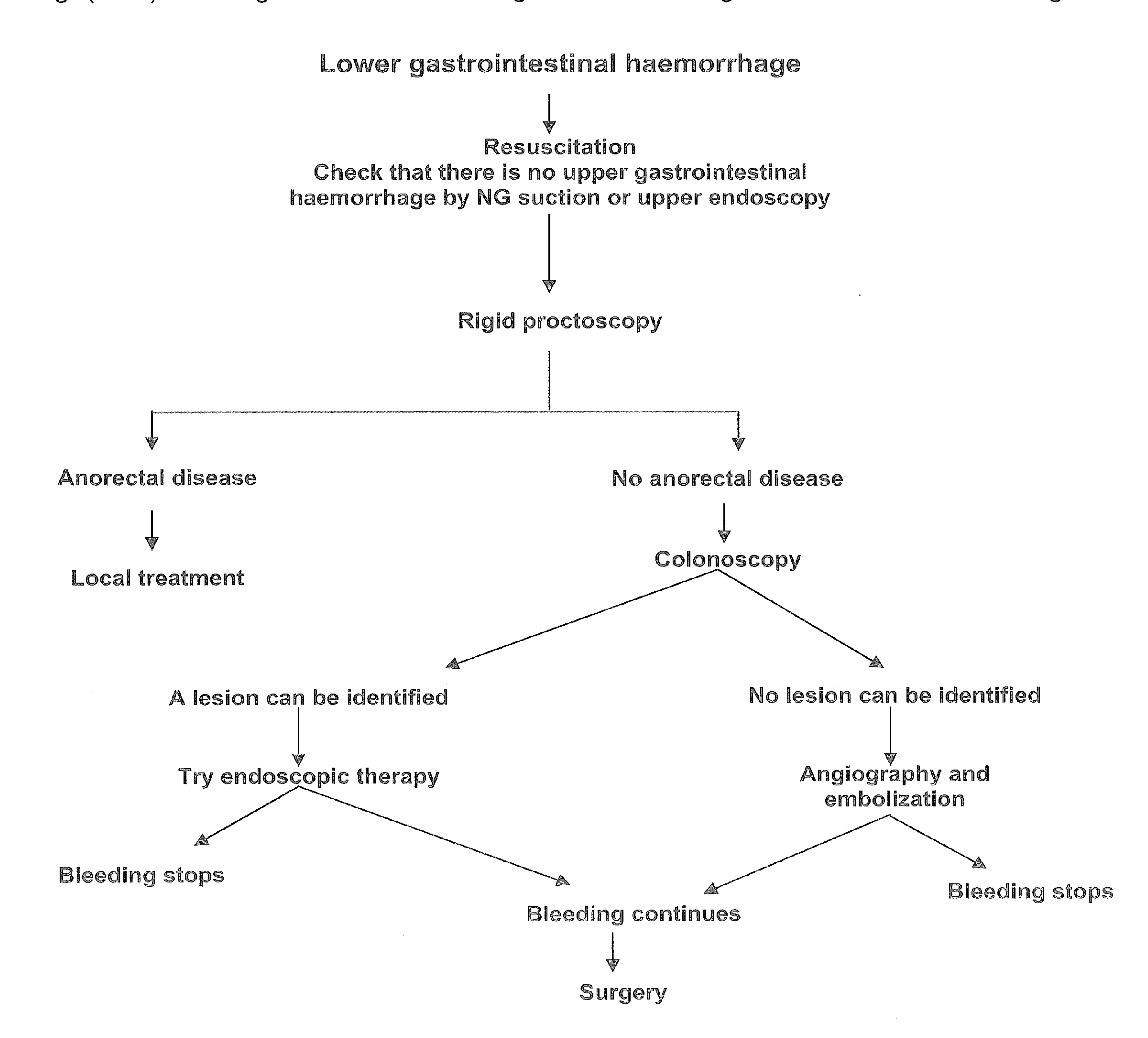
1. Check that the patient does not have upper gastrointestinal bleeding by passing a nasogastric tube or by upper endoscopy.
2. Exclude the causes of generalized bleeding tendency.
3. Stools examination may reveal bilharzial ova or trophozoites of amoebiasis.
4. **Proctoscopy** will reveal internal haemorrhoids.
5. **Sigmoidoscopy**. The rigid sigmoidoscope can reach up to 30 cm from the anal verge while the fibreoptic sigmoidoscope can reach up to 70 cm and so it can diagnose most of the lesions of the rectum, sigmoid colon and descending colon.
6. **Colonoscopy** can visualize the whole colon but it needs proper preparation of the colon by repeated enemas before the procedure. In patients with massive colonic bleeding, the blood will obscure the field and so it is better to postpone the procedure in these situations. Colonoscopy is the investigation of choice for chronic blood loss.
7. **Isotope scans**. The patient's own RBCs are tagged with ^{99m}Tc and then injected intravenously. Abdominal scanning by a gamma camera can identify the site of bleeding.
8. **Angiography**. This invasive investigation is performed when colonoscopy cannot be performed because of massive bleeding or when colonoscopy cannot pinpoint the source of bleeding, e.g., in angiomatous malformations of the colon. Selective catheterization of the superior or inferior mesenteric artery will usually succeed in localizing the site of bleeding provided that the rate of bleeding is 0.2-0.5 ml or more per minute. If angiography succeeds in localizing the source of bleeding, an attempt can be made to stop the bleeding by injection of vasoconstrictors or gel foam through the angiography catheter. Angiography is not an easy investigation and it is not available except in special centres.
9. Contrast radiology. Double contrast barium enema is only justified as an elective investigation in case of chronic blood loss.
10. **Laparotomy**. If all the previous investigations are not available, or failed to pinpoint the site of bleeding, it may be inevitable to proceed to laparotomy in patients with massive bleeding. Colonoscopy can be performed during exploration.

Treatment

1. For massive bleeding start the usual resuscitative measures.
2. Fortunately in the majority of cases, bleeding will stop spontaneously and the surgeon has the time to diagnose and treat the patient electively.

3. If massive bleeding continues, proceed with colonoscopy or angiography according to the available experience and facilities. If angiography succeeds in localizing the bleeding point, an attempt can be made to stop bleeding by injection of vasopressin 0.2 unit/minute or by embolization with thrombin or gel foam. If colonoscopy visualizes an area of vascular malformation (angiodysplasia), bleeding can be stopped by diathermy or laser.
4. If all the previous measures fail to stop bleeding or if the bleeding is massive (blood loss more than 2,5 litres over 48 hours), surgical intervention will have a lower mortality than continued conservative management. If the source of bleeding could be localized preoperatively, segmental resection of the colon would be performed.
5. If there are absolutely no clues as to the source of bleeding, total colectomy may be indicated.

Fig. (40.2) is an algorithm for the management of lower gastrointestinal haemorrhage



Occult bleeding per rectum

This means the passage of a small quantity of blood per rectum that cannot be detected by the gross appearance of the stools.

Causes

1. GIT malignancy.
2. Gastro-oesophageal refiux disease (GORD) and oesophagitis.
3. Peptic ulcer.
4. GIT polyps.

Clinical features and diagnosis

1. Anaemia
    a. Weakness, fatigue, shortness of breath or faintness.
    b. Accidentally discovered anemia
2. Routine screening in certain industries where workers are exposed to GI carcinogens.
3. To examine stools for occult blood, special tests are performed depending on the peroxidase activity of haemoglobin. The patient should avoid the use of a toothbrush, the intake of certain drugs as iron or aspirin, and certain foodstuffs as meat, fish, fresh vegetables and fruits for a few days before the test. A positive test denotes the presence of a source of blood loss of about 20 ml/day.

Postoperative abdominal distension and vomiting

1. Acute gastric dilatation.
2. Paralytic ileus.
3. Intraabdominal abscess, e.g. subphrenic, or pelvic.
4. Peritonitis due to a leaking anastomosis or perforation of a hollow organ.
5. Adhesive intestinal obstruction.
6. Medical causes as uraemia or diabetic keto-acidosis.

Postoperative pyrexia

1. In the first 24 hours mild pyrexia may occur as a reaction to the stress of the operation.
2. In the first few days the commonest cause of pyrexia is respiratory complications, usually atelectasis and pneumonia.
3. Surgical site infection.
4. Deep vein thrombosis.
5. Thrombophlebitis at the site of a peripheral cannula or a central venous catheter.
6. Urinary tract infection especially in the presence of a catheter.
7. Intra-abdominal abscess.

ABDOMINAL WALL AND HERNIA

Abdominal incisions

CHAPTER CONTENTS

Requirements

The requirements of a satisfactory abdominal incision are:

1. Accessibility: The incision should provide good exposure of the diseased area.
2. Extensibility: If needed, it should be possible to enlarge the incision to give more exposure.
3. Safety: The incision should inflict the minimal damage to muscles, blood vessels and nerves. Muscle splitting incisions are preferable than muscle cutting ones.
4. Good cosmetic result, e.g., a transverse suprapubic incision is better looking than a low mid line incision.

Types

Abdominal incisions may be vertical, transverse or oblique (Fig. 41.1).

1. Vertical incisions. Midline, paramedian and transrectal, any one of them may be upper or lower.
2. Transverse incisions. Transverse epigastric, pfannenstiel, and lumbar.
3. Oblique incisions. Subcostal, McBurney's, and Abernathy.

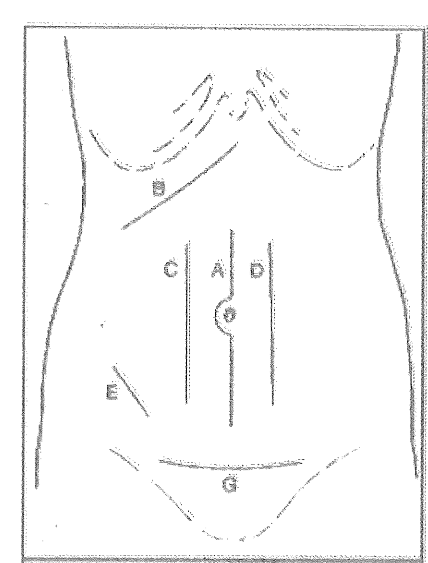


Fig. 41.1. Common abdominal incisions.
A. Midline incision
B. Right subcostal (Kocher's) incision
C. Right paramedian incision
D. D. Left paramedian incision
E. McBurney's incision
G. Pfannenstiel's incision

Vertical incisions

1. **Midline incision**. This incision passes through the linea alba and the two recti are retracted apart. It is a good exploratory incision allowing access to both sides of the abdomen. It is quick and can be enlarged freely. The incision in the linea alba should be closed by a non-absorbable suture material as prolene to avoid incisional hernia and burst abdomen.
2. **Paramedian incision**. This is upper or lower, right or left. The skin incision is one inch from the middle line, the anterior rectus sheath is incised vertically, and the rectus muscle is displaced laterally to preserve the vessels and nerves supplying the rectus muscle from the lateral side. Then the posterior rectus sheath and peritoneum are incised as one layer. This incision is safe and the healing power is strong. It is readily extensible and can give exposure to any abdominal organ. Its only disadvantage is that it is time consuming and is, therefore, not recommended in emergencies.

3. **Paramedian transrectal incision**. The incision is similar to the classic paramedian incision but the rectus muscle is split longitudinally in the same line of incision in the anterior rectus sheath. It is done in emergencies because it is quick, but the scar is not as strong as that of the paramedian incision due to devitalization of the medial part of the rectus muscle. The incision is most useful in children because atrophy is compensated for during growth.

Transverse incisions

Upper transverse epigastric and low transverse suprapubic (Pfannenstiel). In Pfannenstiel incision the anterior Thectus sheath is cut transversely, and then the two recti are separated. Finally the peritoneum is opened.

Advantages

- The scar is cosmetic as the wound lies in Langers lines.
- The muscular tension on the suture line is minimal, and the patient can safely cough in the postoperative period.

Disadvantages

- It is time consuming.
- Excessive bleeding.

Oblique incisions

1. Subcostal incision. On the right side it is called Kocher's incision. It is a muscle cutting incision, one inch below and parallel to the costal margin. In its medial part the rectus muscle and the anterior and posterior rectus sheaths are divided. Laterally, the three abdominal muscles may be divided.
   - **Advantages**: Very good exposure to the biliary apparatus or the spleen especially in obese patients with wide costal angle.
   - **Disadvantages:**
     - Muscle cutting.
     - It is not an exploratory incision.
     - It is not extensible.
     - Injury to the eighth, ninth or tenth intercostal nerves with subsequent hypoesthesia or paralysis of a part of the abdominal wall may occur.
2. **McBurney's incision**. It is a muscle splitting incision, very commonly used for appendicectomy. The incision is about 5-6 cm and is perpendicular to a line passing from the anterior superior iliac spine to the umbilicus at the junction of its outer third with the inner two thirds. The external oblique aponeurosis is opened in the same direction of the skin incision, while the internal oblique and transversus abdominis muscles are split in the direction of their fibres.
   - **Advantages:**
     - Good exposure to the caecum and appendix.
     - Safe incision as the muscles are not cut.
     - The incision is cosmetic particularly if the skin incision is made transversely (Lanz incision).
   - **Disadvantages**: It is not an exploratory incision. It is not recommended if the diagnosis of appendicitis is not sure. The incision can be extended upwards or downwards, but it will then be a muscle cutting incision.

Precautions during closure of an abdominal incision

1. A non-absorbable monofilamentous suture material, e.g., polypropylene (prolene) is used.
2. The linea alba or the anterior rectus sheath is the strongest layer. Its sound closure is essential for safety of wound closure. Wide bites (1 cm) are taken from the edge of the fascial incision. The suture length to the wound length should be 4:1.
3. The sutures should not be under tension to avoid ischaemia of the wound.
4. If there is a peritoneal defect, leave it as the peritoneum will regenerate in a few days.


Fig. 41.2. Burst abdomen after an upper midline abdominal incision.

Complications of abdominal incisions

1. Haematoma. This may be due to a bleeding tendency in the patient but far more commonly due to careless surgical haemostasis. It causes dull aching pain in the wound which is indurated and may be discoloured. If the haematoma is small it can be left for spontaneous absorption but if enlarging or of a large size, it should be evacuated to avoid secondary infection.
2. **Infection (Chapter 7).**
3. Wound disruption (burst abdomen). This is a serious complication which may lead to an incisional hernia or may even cause mortality,
4. Incisional hernia. The causes of incisional hernia are the same as burst abdomen, and as a matter of fact many patients with incisional hernia had partial disruption of the deeper layers of the abdominal wound during the immediate or early post-operative period.
5. **Desmoid tumour**.

Burst abdomen

Predisposing factors

Pre-operative factors

1. Obesity.
2. Factors which cause poor healing as malnutrition, cirrhosis, diabetes mellitus, jaundice and corticosteroid intake (Chapter 1).
3. Patients with respiratory problems as chronic bronchitis, bronchial asthma and chronic obstructive lung disease.
4. The nature of the primary disease for which the operation was performed, e.g., patients with abdominal malignancy are usually malnourished and patients with peritonitis will have abdominal distension and wound sepsis

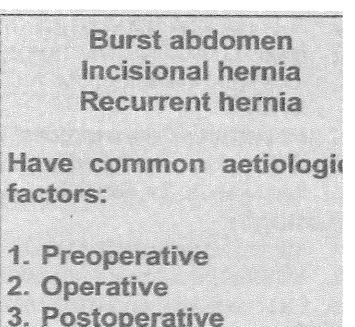
Operative factors

1. Muscle cutting incisions are more liable to burst than muscle splitting ones.
2. Vertical incisions have a higher incidence of burst than transverse ones.
3. Rough surgical technique with excessiye trauma to the muscles, blood vessels and nerves.

4. Use of absorbable sutures in the closure of the aponeurotic layer of an abdomi wound. Non-absorbable sutures as polypropylene are recommended. Good bi should be taken on either side of this layer.
5. Insertion of drainage tubes through the main wound.

Post-operative factors

1. Poor recovery from anaesthesia leading to strong coughing.
2. Persistent increase in intra-abdominal pressure due to repeated coughing, vomiting, hiccough or abdominal distension.
3. Haematoma of the wound.
4. Surgical site infection is the most important factor. The tissues become friable due to collagen lysis allowing the sutures to cut through them.



Burst abdomen
Incisional hernia
Recurrent hernia

Have common aetiologi factors:

1. Preoperative
2. Operative
3. Postoperative

Types

- **Partial**. The deep layers burst but the skin is intact. This actually produces an incisional hernia.
- Complete.
- If the intestine prolapses out of the wound, it is called evisceration.
- If the intestine is retained inside the abdomen, it is called dehiscence (Fig. 41.2).

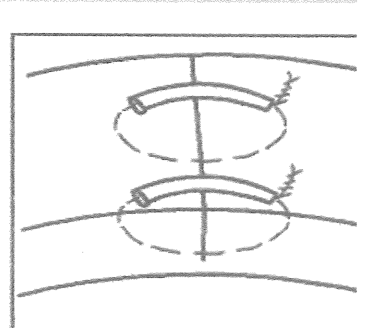


Fig. 41.3. Retention sutures clude the full thickness of dominal wall.

Clinical features

- A warning sign to the occurrence of burst is "the red sign" where a serosanguinous discharge soaks the dressing. It is due to strangulation of a piece of omentum or a loop of bowel which is prolapsed through a defect in the muscles.
- The burst usually occurs on **6th** to the **8th** day post-operatively.
- The patient often feels as if something gives way.
- Symptoms of intestinal obstruction may be present.

Treatment is by urgent surgical closure.

- Cover the prolapsed bowel by a sterile dressing.
- Insert a nasogastric tube and start an IV infusion.
- In theatre under general anaesthesia the protruding loops of bowel are washed w saline and returned to the abdominal cavity. The omentum is spread over t intestine. The abdominal wall is closed as one layer by through-and-through sutur using strong non-absorbable suture material, e.g., polypropylene. These are call "retention sutures" (Fig. 41.3) as they are retained for at least three weeks. Ca should be taken not to puncture a loop of bowel.
- Antibiotics are prescribed and an abdominal binder is recommended.

Minimal access surgery

The last decade of the twentieth century witnessed the evolution of what is kno as "minimal access surgery". Many operations can now be performed either through t natural body orifices using fibre-optic endoscopy, or through fine stabs that are used introduce rigid endoscopes into the peritoneum, pleura, and joint cavities.

Laparoscopy

For abdominal surgery, laparoscopy has developed to such an extent that many operations can now be performed using this technique.

Frequently performed laparoscopic operations:

1. Cholecystectomy.
2. Appendicectomy.
3. Inguinal hernia repair.
4. Bariatric surgery.
5. Fundoplication for gastro-oesophageal reflux.
6. Gynaecologic operations.
    a. Tubal ligation.
    b. Tubal adhesiolysis.

Steps

1. General anaesthesia.
2. Insufflation of peritoneal cavity with CO_2 using a Veress needle that reduces the possibility of pucturing viscera during its introduction. Gas in the peritoneal cavity makes a space between the anterior abdominal wall and the viscera. This space allows visualization of organs and manipulation of instruments.
3. A trocar and cannula are inserted, usually at the umbilicus. The trocar is removed and the cannula (port) is used to introduce the telescope. This telescope is connected to a video camera that displays its image on a monitor and allows the surgeon and assistants to see the interior of the abdominal cavity.
4. Inspection of the peritoneal cavity.
5. Other ports are inserted under direct vision through the abdominal wall to allow the introduction of instruments for dissection, coagulation, retraction and cutting.

Advantages

1. Minimal postoperative pain.
2. Minimal impairment of pulmonary functions.
3. Fast recovery and early return to normal activities.
4. The ability to visualize and explore the whole abdominal and pelvic organs.
5. Video recording of the operative procedures with obvious educational advantages.
6. Better appearance and decreased wound problems as dehiscence or infection.

Drawbacks

1. The need for well-trained surgeons.
2. High cost of the equipment.
3. Postoperative shoulder pain, which is caused by irritation and stretching of the diaphragm by CO_2.

Conversion of laparoscopic surgery into the conventional oen surgery is indicated in the following situations considering that the safety of the patient is the absolute priority:

1. Equipment failure.
2. Dense adhesions or anatomical abnormalities precluding safe performance of the procedure.
3. Uncontrolled bleeding.
4. Accidental injuries requiring open repair.

Diagnostic laparoscopy

Is rapidly gaining popularity in certain situations e.g.

- Determination of the cause of acute lower abdominal pain e.g. acute pelvic appendicitis or torsion of an ovarian cyst.
- Determination of the extent of malignant disease e.g. small liver secondaries or peritoneal nodules.
- In blunt abdominal trauma to detect the exact injuries.

Diseases of the abdominal wall

Diseases of the umbilicus

1. **Umbilical fistula**
   a. Faecal fistula is either congenital from a patent vitello-intestinal duct (Fig. 41.4), traumatic, inflammatory from tuberculosis of small intestine or malignant from carcinoma of the transverse colon ulcerating through the umbilicus.
   b. Urinary fistula is either congenital from patent urachus or is rarely acquired.
   c. Biliary fistula at the umbilicus is very rare and may be due to operative bile duct injury during cholecystectomy.

Fig. 41.4. Patent vitello-intestinal duct that causes a congenital fecal fistula at the umbilicus.

2. **Umbilical sinus** discharges pus and is due to either an abdominal wall abscess or umbilical infection. Pilonidal sinus of the umbilicus is rare, but it leads to persistent discharge.
3. **Umbilical stone** may form due to chronic inflammation of umbilicus or from umbilical granuloma. The stone should be removed, the granuloma is excised by diathermy and antiseptics applied.
4. **Umbilicat polyp** is due to persistence of the umbilical extremity of the vitello-intestinal duct, which becomes everted outwards. The epithelial surface undergoes irritative hyperplasia from friction leading to the formation of a polypoid mass at the bottom of the umbilicus (Fig. 41.5). It should be excised.

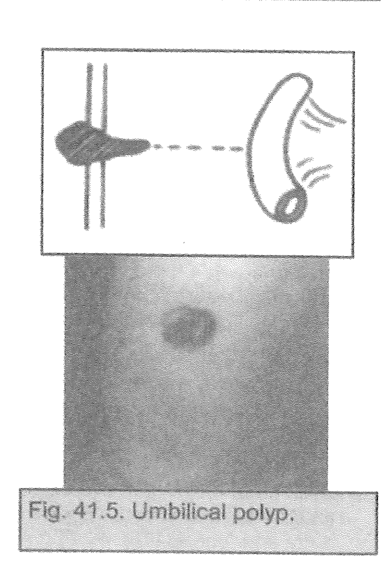


Fig. 41.5. Umbilical polyp.

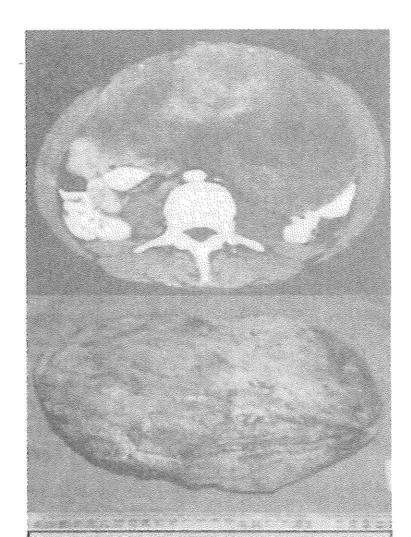
5. **Umbilical granuloma**. This is a mass of granulation tissue due to chronic infection of the umbilical scar. It should be curetted and then cauterized by silver nitrate.
6. **Umbilical hernia**.
7. **Tumours of the umbilicus**.
   a. Squamous cell carcinoma is rare, It gives metastasis to axillary and inguinal lymph nodes on both sides.
   b. Secondary carcinoma nodules may be present at the umbilicus due to spread from carcinoma of stomach, pancreas, liver or breast.

Desmoid tumour

Pathology

- The nature of this tumour is not exactly known. It is considered by some authorities as a locally malignant fibrosarcoma,

- It arises from the anterior or less commonly the posterior rectus sheath, or from the anterior abdominal wall muscles. It may occur on top of scars or incisions.
- A desmoid tumour may be associated with intestinal polyposis (Gardner's syndrome).
- Gross appearance. The tumour is a non-encapsulated, slowly growing swelling that infiltrates the surroundings structures. Cut section is pinkish white.
- Microscopically it formed of cellular fibrous tissue.


Fig. 41.6. Huge desmoid tumour of anterior abdominal wall as shown on CT scan. The lower picture shows the tumour after wide excision.

Clinical features

- A desmoid tumour usually affects females about the age of 40 years.
- The patient presents by a painless, hard, ill-defined, slowly growing mass of the abdominal wall with a nodular surface.

Treatment is by excision with a safety margin of at least one inch (Fig. 41.6) and reconstruction of the defect by flaps of fascia or synthetic mesh. Recurrence is very common if the tumour is not adequately excised.

Haematoma of the reCtus sheath

This is usually due to trauma causing rupture of the inferior epigastric vessels.

Clinically there is pain, tenderness and swelling over the rectus muscle.

Treatment. If the haematoma is large, evacuation of the haematoma and ligation of the epigstric vessels are performed.

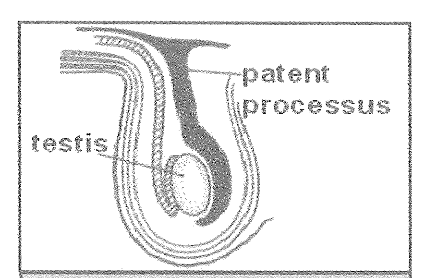


Fig. 41.7. A patent processus vaginalis causes a congenital inguinal hernia.

General principles of external abdominal hernias

Definition

An external abdominal hernia is a protrusion of a viscus or part of a viscus, usually within a peritoneal sac, through a defect in the abdominal wall.

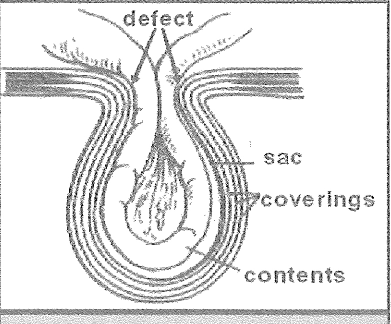


Fig. 41.8. Components of a hernia.

Aetiology

Congenital (preformed) sac

- Unobliterated processus vaginalis causes congenital inguinal hernia (Fig. 41.7).
- Unobliterated physiological umbilical hernia of foetus causes congenital umbilical hernia (exomphalos).

Acquired causes

- Raised intra-abdominal pressure due to chronic cough, straining at micturition or stools, heavy work, obesity, or a huge abdominal swelling (splenomegaly or pregnant uterus).
- Weak abdominal wall due to obesity, senility, debility, pregnancy, weak scar, and damaged nerve supply of the muscles.

Components

A hernia consists of three parts, which are the sac, the contents and the coverings of the sac. The contents pass out through a defect in the abdominal wall (Fig. 41.8).

1. **Sac**. This is the peritoneal pouch which bulges out through the abdominal wall defect. It has a neck (its junction with peritoneum), body and fundus (Fig. 41.9).
2. **Contents**. Any abdominal viscus can protrude out into the sac except the pancreas. The usual contents are intestine, omentum or both. Reduction of a hernia means reduction of its contents into the peritoneal cavity while the empty sac remains in place.

   Special contents
   - Richter's hernia. The content is a part of the bowel circumference (Fig. 41.10). This type is common in a femoral hernia.
   - Littré's hernia. The content is Meckel's diverticulum.
   - Maydl's (W) hernia. It contains two loops of intestine while an intermediate loop lies in peritoneal cavity (Fig. 41.11).
   - Urinary bladder: A part of the bladder may protrude into an inguinal or femoral hernia. It usually protrudes along the inner side of the sac lying outside it, forming a sliding hernia.
3. **Coverings.** These are the structures that are stretched over the sac.

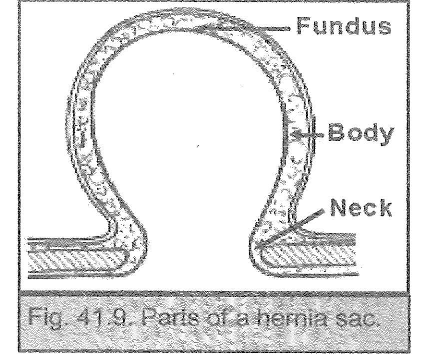
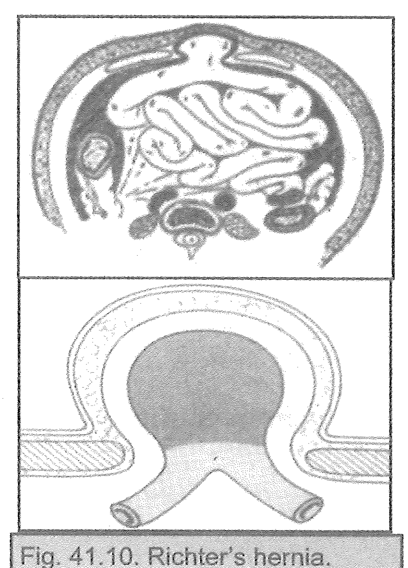
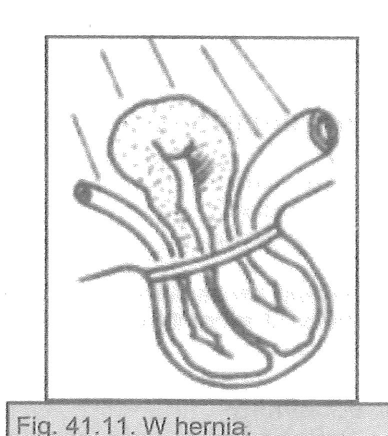


Fig. 41.9. Parts of a hernia sac.


Fig. 41.10. Richter's hernia.


Fig. 41.11. W hernia.

Diagnosis

When a clinical diagnosis of a hernia is suspected, the following questions need to be answered:

1. Is this swelling a hernia? Hernia is a swelling that is characterized by:
   a. The swelling is present at one of the anatomical sites of a hernia e.g. inguinal, femoral, umbilical, epigastric or incisional.
   b. It has an expansible impulse on cough (except if strangulated).
   c. It is reducible (except if irreducible, obstructed, or strangu lated).
   d. By transillurnination it is opaque except in infants.
2. **Which type**? This depends on the exact site, special features and special tests.
3. **What are the contents?** These are usually intestine or omentum. Table 41.1 shows the clinical differences between them.

Table 41.1. Clinical differences between enterocele and omentocele

	Intestine (enterocele)	Omentum (omentocele)
Consistency	Soft	Doughy
Gurgling	Occurs during reduction	None
Ease of reduction	First part is more difficult to reduce than the last	Last part is more difficult to reduce
Percussion	May be reasonant	dull

4. Is it complicated? Complications are discussed below.
5. Is there any cause for raised intra-abdominal pressure or weak abdominal wall?
   History and examination of chest, abdomen, urinary tract and anus are essential. Such causes should be treated before hernia operation. Weak abdominal muscles presents by either:
   - Multiplicity of hernias (Fig. 41.12).
   - Divarication of recti.
   - Bulge of lower abdomen on straining (Malgaigne bulge, Fig. 41.12).
6. Is there any other hernia?
7. Is the patient fit for surgery?
   History and examination for cardiac complications, respiratory problems, hypertension and diabetes, should be completed before surgery.



Fig. 41.12. This elderly patient has weak abdominal muscles. He has an epigastric hernia and a right inguinal hernia. In addition he has Malgaigne bulge of lower abdomen.

Complications of a hernia are those of its contents.

The most serious is strangulation.

Strangulated hernia requires urgent surgery

Treatment

1. **Operation is always advised** to avoid complications of hernia and to prevent its progressive enlargement which widens the defect and weakens the musculoaponeurotic layers around the defect. Uncomplicated hernias are repaired by elective surgery after eradication of the predisposing factors.
2. Truss to control herniation, is better avoided, but may be used for extremely unfit patients (Fig. 41.13).

The standard treatment of external abdominal hernias is SURGERY.

There are three main types of hernia operations:

- **Herniotomy** (herniectomy) means excision of the sac. It is either done alone or as a part of heniorrhaphy or hernioplasty.
- **Herniorrhaphy** entails heniotomy and repair of the defect by approximation of the local tissues.
- **Hernioplasty** entails herniotomy and closure of the defect without tension using imported material, which are not from the vicinity of the defect. This material may be tissues from distant parts of the body, e.g., fascia lata or synthetic material. The usual example is the use of synthetic mesh, e.g., polypropylene No-tension repair by mesh hernioplasty is popular nowadays as it has the lowest incidence of hernia recurrence.

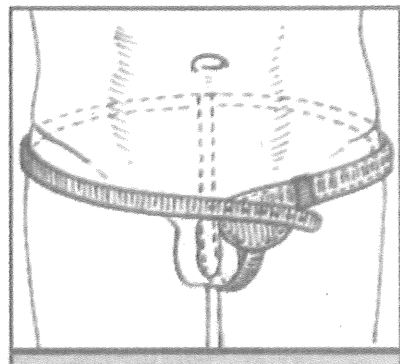


Fig. 41.13. A truss is rarely indicated.

Complications

1. Irreducibility.
2. Obstruction
3. Strangulation
4. Inflammation
5. Hydrocele of a hernia sac.
6. Torsion of omentum.

In general hernia complications are those of the contents, i.e., irreducibility of a hernia means that its contents are irreducible and a strangulated hernia means that its contents are strangulated (its blood supply is cut-off).

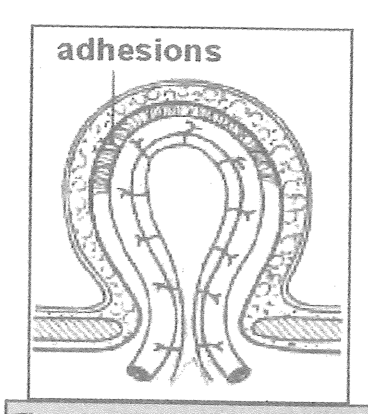


Fig. 41.14. Irreducible hernia because of adhesions between the contents (intestine) and the sac.

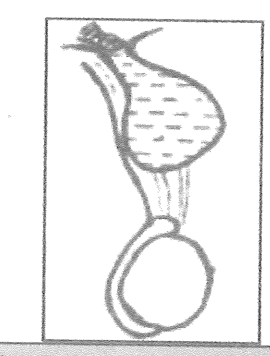
Irreducibility

Definition. Irreducibility means failure to return the contents into the abdomen.

Causes

- Adhesions that form within the sac either between the contents and the sac (Fig. 41.14) or between the contents themselves.
- Protrusion of more omentum within the sac. Irreducibility predisposes to obstruction and strangulation and so operation is essential.

Irreducibility without other symptoms of obstruction is almost diagnostic of an omentocele.


Fig. 41.15. Hydrocele of a hernia sac.

Obstruction

- This occurs in irreducible hernias due to occlusion of the intestinal lumen from without or from within.
- In a purely obstructed hernia the blood supply is unaffected.
- There are symptoms of intestinal obstruction as vomiting, distension, colics, and constipation. The picture simulates strangulation but is less severe.
- Locally the hernia becomes distended, irreducible, but it is still soft.
- Distinction between obstruction and strangulation in hernias may be difficult. It is, thus, safer to treat it as strangulation and early surgery should be performed.

In any patient with intestinal obstruction all hernia orifices should be examined so as not to miss a strangulated or an obstructed hernia.

A femoral hernia is the most frequently missed.

Strangulation

This is the most serious complication of a hernia. It will be discussed separately.

Inflammation

- This is an uncommon problem in a hernia, ilt means inflammation of the contents.
- Causes
  - Rough taxis.
  - Ill-fitting truss.
  - Spontaneous inflammation of contents (appendix, fallopian tube or ovary).

- Clinical features. The hernia is tender but not tense, and the overlying skin is red and oedematous.
- Treatment. Operation is essential, as strangulation cannot be excluded.

Hydrocele of the hernia sac
- This occurs in narrow-necked sacs if the contents return to abdomen and fail to descend in the sac again. The neck becomes occluded by omentum and serous fluid collects in the sac.
- Clinically there is a cystic swelling in the upper part of the spermatic cord.

Torsion of the omentum.

Stangulated hernia

Strangulation is the most serious hernia complication.

Definition

Strangulation of a hernia means constriction of contents leading to interruption of their blood supply. If not relieved, gangrene may occur within a few hours.

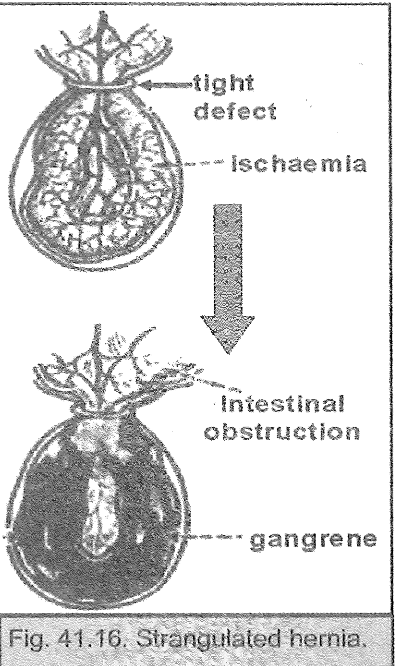


Fig. 41.16. Strangulated hernia.

Incidence
- It varies according to the type of hernia. It is estimated to be about 2-4% in inguinal, 25-30% in femoral 15-20% in paraumbilical and 3-5% in incisional hernias.
- Although the incidence of strangulation is higher in femoral hernias, yet, strangulated inguinal hernias account for more than 50% of all strangulated external hernias.
- Strangulation can occur at any age and is commoner after prolonged use of a truss.

Causes of strangulation
- Extrusion of new contents following straining.
- Repeated attempts at reduction, producing oedema.

Pathology (Fig. 41.16)

The constricting agent may be:
- A resistant structure outside the sac as the superficial or deep inguinal ring or the Gimbernats ligament.
- The neck of the sac.
- Bands of adhesions within the sac.

Consequences
- If the contents are intestines, the intestine proximal to the strangulated loop will be obstructed with progressive distension and hyperperistalsis. The intestine distal to the strangulated loop will be collapsed.
- The strangulated loop will suffer the following sequelae:
    - Impeded venous return. The strangulated intestine becomes congested and distended with accumulating gas and fluid (congestion increases secretions and decreases absorption). Increased congestion causes haemorrhage in the wall of intestine, into its lumen and from its surface into the sac.

- Later, the arterial supply becomes impaired and the devitalized intesti exudes its contents (fluid, blood, bacteria) through its wall, to the sac whi thus contains dark highly toxic fluid.
- Finally, gangrene occurs. It starts at the ring of constriction. It then affects t antimesenteric border (convexity) of the loop. Perforation may occur at the sites. Later, gangrene affects the whole loop and its mesentery.
- Peritonitis is the terminal event, as infection spreads from the sac to t peritoneum. Neglected cases die from septic shock and dehydration.

Clinical features

Symptoms

1. Acute pain in the hernia.
2. Sudden enlargement. The hernia is bigger.
3. Irreducibility. The hernia, which used to disappear on lying down or by pressing now fails to reduce. Sometimes a hernia presents for the first time by strangulation.
4. If the hernia contains intestine the patient develops symptoms of intestin obstruction, which are colicky abdominal pain, vomiting, absolute constipation and distension. Intestinal obstruction is not present if the content is omentum, part of bowel circumference (Richter's hernia), or Meckel's diverticulum (Littre's hernia).

A strangulated hernia is commonly associated with intestina obstruction. It does not produc obstruction if:
1. Contents are omentum.
2. Richter's hernia.
3. Littré's hernia.

General examination usually shows no abnormality except in neglected cases where hypovolaemic, or even septic shock, sets in.

Strangulation causes:
1. Acute pain in the hernia.
2. Hernia becomes tense, tender, irreducible with no impulse on cough.

Local examination. The hernia is:

1. Tense.
2. Tender.
3. Irreducible
4. There is no impulse on cough.

Treatment

The treatment of strangulated hernia is urgent surgery.

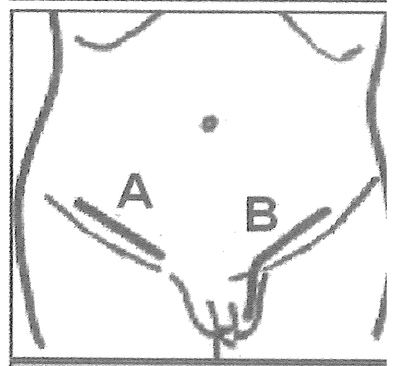


Fig. 41.17. Incisions for inguina hernia.
A. Uncomplicated hernia.
B. Strangulated hernia. The incision extends to the scrotum to allow opening the fundus and evacuating toxic contents of the sac at an early stage o operation.

Preoperative preparation (as in any case of intestinal obstruction):

1. Nasogastric tube suction.
2. IV infusion of lactated Ringer's solution to correct hypovolaemia.
3. IV broad spectrum antibiotics to guard against septicaemic shock.

Steps of the operation

1. The incision should be planned to expose the fundus of the sac (Fig. 41.17).
2. The fundus of the sac is opened at first to evacuate the toxic fluid, which is full of organisms.

3. The constricting ring should then be divided. A grooved director or the left finger is inserted inside this ring and its coverings are divided from outside until the constriction is relieved.
4. The contents are pulled out and examined. Pulling out on intestinal loops in the hernia is important so as not to miss a strangulated Maydl's hernia (Fig. 41.11). Table 41.2 shows the differences between viable and non-viable intestine at operation.

Table 41.2. Differences between viable and non-viabl intestine at operation

Viable	Non-viable
Normal luster	Lusterless
Pink	Grey or black
Pulsating mesenteric arteries	Not pulsating
Bleeds if injured	Does not bleed
Firm	Flabby & thin
Contracts if pinched	No response

5. Dealing with the contents.
   a. The omentum is always excised.
   b. Viable intestine is returned to the abdomen.
   c. Gangrenous small intestine needs resection and primary anastomosis.
   d. Gangrenous colon is treated by resection of the gangrenous segment and colostomy. Anastomosis of an unprepared colon is better avoided. In these cases elective anastomosis after colon preparation is done after a few weeks.
   e. If the intestine looks suspicious, warm packs are applied and the patient is given pure oxygen for a few minutes. Decision is then taken whether the intestine is viable or gangrenous.
6. Repair of the hernia defect using polypropylene sutures as this suture material is inert and does not cause an inflammatory reaction.
7. Subcutaneous drains are usually needed.

Postoperative management

1. Nasogastric tube suction and IV fluids are continued until intestinal sounds are audible (usually 2-3 days).
2. Prophylactic antibiotics.
3. Drains are removed when they stop discharging (usually 5-7 days).

Sliding hernia

This is a hernia where a viscus forms a part of the wall of the sac. The commonest are the bladder, caecum (Fig. 41.18) and sigmoid colon. It is common in old-standing hernias, in males and in old age.

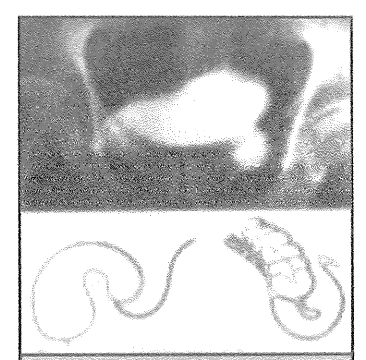


Fig. 41.18. Sliding hernia. The cystogram shows a sliding part of the bladder. Note that in a this type of hernia the sliding viscus forms part of the sac wall.

Clinical features

- There is usually a longstanding hernia in an obese elderly man.
- The hernia is usually complete oblique inguinal hernia.
- After reduction of the contents there is still fullness at the site of the hernia, i.e., the hernia is partially reducible.

- There are urinary symptoms in case of a sliding bladder, e.g., pressing the hernia causes a desire to void, double micturition, and reduction of hernia size after micturition.

Treatment

Do not try to dissect the sliding viscus from the sac as this may lead to devascularization or injury of the viscus.

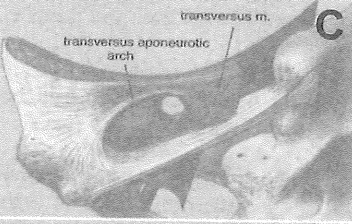
Free the sliding sac and viscus well from the surrounding structures and push them back behind the transversalis fascia which is then repaired. Then perform a strong repair to the inguinal canal using a mesh if required.

Surgical anatomy of the inguinal region

Layers of abdominal wall in the inguinal region

The layers of the abdominal wall in the inguinal region include the following from superficial to deep aspects (Fig. 41.19).

1. **Skin.**
2. **Superficial fascia** is composed of a superficial fatty layer (Camper' fascia) and a deep membraneous layer (Scarpa's fascia). The latter allows free gliding of fat over the muscles. The superficial fascia of the abdomen does not communicate with the corresponding layer of the thigh due to the presence of an inguinal crease where the skin is attached to deep fascia, one inch below the inguinal ligament. There is no deep fascia in the abdominal wall to allow for free respiratory movements, gastric fullness and pregnancy.
3. **External oblique muscle arises** by fleshy digitations from the lower 8 ribs. Its fibres run downwards and forwards, and become aponeurotic from the level of the umbilicus down to its free lower border which is infolded and thick and is called the inguinal (Poupart's) ligament. This stretches between the pubic tubercle medially and the anterior superior iliac spine laterally. The free posterior margin of the inguinal ligament fuses with lower ends of fascia transversalis and iliaca. In its medial fourth the infolded part of the inguinal ligament is thick and is attached to the iliopectineal line of the pubic bone to form the lacunar (Gimbernat's) ligament. Thus, the infolded surface of the inguinal and lacunar ligaments makes a floor for the spermatic cord. The inguinal ligament is convex downwards, being attached to the deep fascia of the thigh (fascia lata).
4. **Internal oblique muscle arises by** fleshy fibres from the lateral half (or two thirds) of the reflected surface of the inguinal ligament. Its lower border covers the deep inguinal ring and the beginning of the spermatic cord. It arches horizontally above the cord and fuses with the lowest fibres of transversus muscle to form the conjoint tendon which passes vertically downwards behind the cord to be inserted into the pubic tubercle and iliopectineal line.



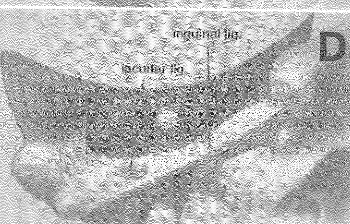


Fig. 41.19. Anatomical layers in inguinal region, except skin and superficial fascia.
A. External oblique, B. Internal oblique, C. Transversus abdominis, D. Fascia transversalis.

5. **Transversus abdominis**. The lower part arises by fleshy fibres from the lateral third of the inguinal ligament and arches horizontally higher than the internal oblique to be inserted through the conjoint tendon in the pubic tubercie and iliopectineal line.
6. **Transversalis fascia** is a thin but strong fascial layer that lies in front of the peritoneum. It is the most important defense against hernia formation. The lower part of the fascia transversalis is thickened forming the iliopubic tract which runs just above and parallel to the inguinal ligament. The upper part of the fascia transversalis is also thickened forming the arch of the transversus abdominis which lies at the lower border of the transversus muscle.

Inguinal canal (Fig. 41.20)

Development

This canal is developed by the passage of the testis from the abdomen to the scrotum.

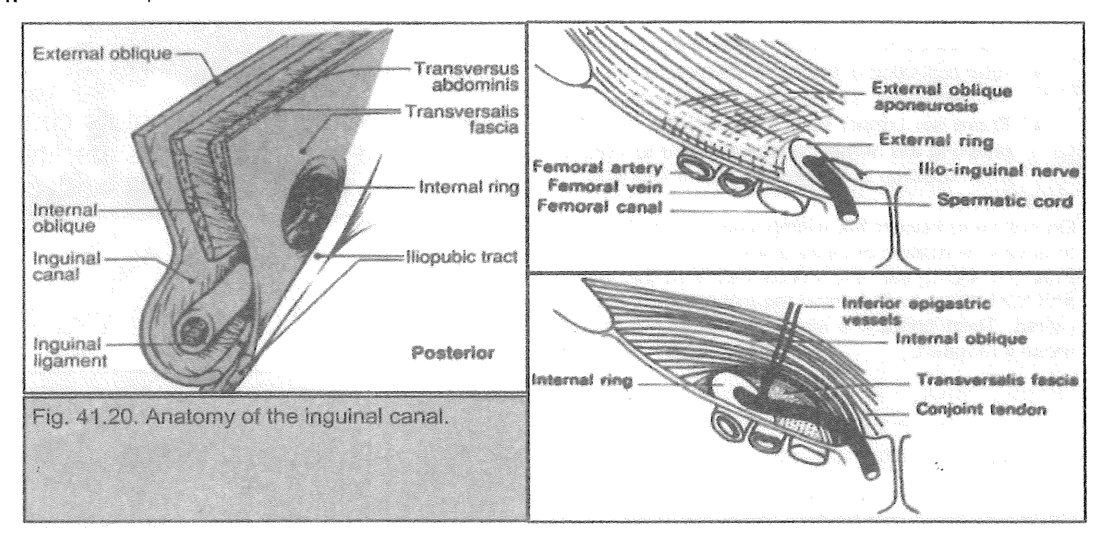


Fig. 41.20. Anatomy of the inguinal canal.

Dimensions and site

In adults the canal is 1.5 inches (4cm) long.

It extends obliquely downwards, medially and forwards in the lower and lateral part of the anterior abdominal wall from the deep to the superficial inguinal rings (above the medial half of the inguinal ligament).

Contents

The inguinal canal contains the sperrnatic cord (round ligament in female) and the ilioinguinal nerve.

Inguinal rings

Deep inguinal ring. This is an opening in the transversalis fascia 1/2 inch above the midinguinal point (midway between the symphysis pubis and the anterior superior iliac spine), The inferior epigastric vessels run medial to it. The deep inguinal ring is covered anterioily by the lower border of the internal oblique muscle.

Superficial inguinal ring. This is a triangular opening in the external oblique aponeurosis that is situated half an inch above and medial to the pubic tubercle. It is bounded by the pubic crest below and the medial and lateral crura which are joined by intercrural fibres. Normally it does not admit the tip of the little finger. It is an exit for the spermatic cord and the ilioinguinal nerve. It is backed by the conjoint tendon.

Boundaries of the inguinal canal. These constitute the relations of the spermatic cord.

- Posteriorly
  - Fascia transversalis.
  - Inferior epigastric vessels laterally.
  - Conjoint tendon medially.
- Anteriorly
  - External oblique aponeurosis.
  - Lower part of internal oblique muscle laterally.
- Superiorly
  - Conjoint muscles (internal oblique and transversus).
- Interiorly
  - Infolded surface of the inguinal ligament with the upper surface of the lacunar ligament medially.

Spermatic cord

Course

The cord begins just deep to the deep inguinal ring by gathering of its constituents. It enters the deep ring, traverses the inguinal canal and exits from the superficial ring. It then passes down in front of the pubic bone, crosses the scrotal neck and enters the scrotum where it is attached to the top and back of the testis.

Constituents

1. Vas deferens.
2. Testicular artery.
3. Pampiniform plexus of veins (testicular veins).
4. Artery of the vas.
5. Testicular lymphatics.
6. Genital branch of genito-femoral nerve.
7. Testicular autonomic nerves.
8. Vestige (remnant) of processus vaginalis, which is anterolateral to the vas and the vessels.

All the structures of the cord are embedded in loose fat and areolar tissue.

Coverings

The spermatic cord has three coverings which are derived from the penetrated layers of the abdominal wall during testicular descent. These covehngs surround the testis and the cord like 3 sockets.

1. The internal spermatic fascia is derived from transversalis fascia at the deep ring.
2. The cremasteric muscle and fascia are derived from the lower border of the internal oblique muscle as it overlies the deep ring. The cremasteric muscle is supplied by the genital branch of genitofemoral nerve and it acts to elevate the testis.
3. The external spermatic fascia is derived from the external oblique aponeurosis at the superficial ring.

Anatomical basis of inguinal hernia

The inguinal region is a weak spot in the anterior abdominal wall due to the following causes:

1. The muscles of the abdominal wall in the inguinal region are aponeurotic and, therefore, this area is weaker than the fleshy parts of the abdomen.
2. The internal oblique and transversus abdominis muscles arch up to form the roof of inguinal canal.

3. The spermatic cord passes between the muscles and adds more weakness to the inguinal area.

Protective mechanisms

1. Obliquity of the inguinal canal.
2. The transversalis fascia although thin, is a strong layer that supports the posterior walL
3. Weak parts of the canal are supported by strong structures. The deep inguinal ring is reinforced by condensation of the transversalis fascia and by the fleshy fibres of lower part of internal oblique in front of it. The superficial inguinal ring is supported by the conjoint tendon posteriorly.
4. Shutter mechanism. During contraction of the abdominal muscles, the lower border of the conjoint tendon straightens and descends downwards toward the inguinal ligament, thus closing the posterior wall.
5. Valvular mechanism. Contraction of the external oblique muscle tightens its aponeurosis and narrows the superficial inguinal ring.
6. Cremasteric mechanism. Contraction of the cremasteric muscle plugs the superficial inguinal ring and causes bulging of the spermatic cord in the middle third of inguinal canal leading to its obliteration.
7. Contraction of the transversus abdominis muscle pulls and tenses the edges of the internal ring.

The different types of groin hernias are shown in relation to anatomical structures of the area in Figure 41.21.

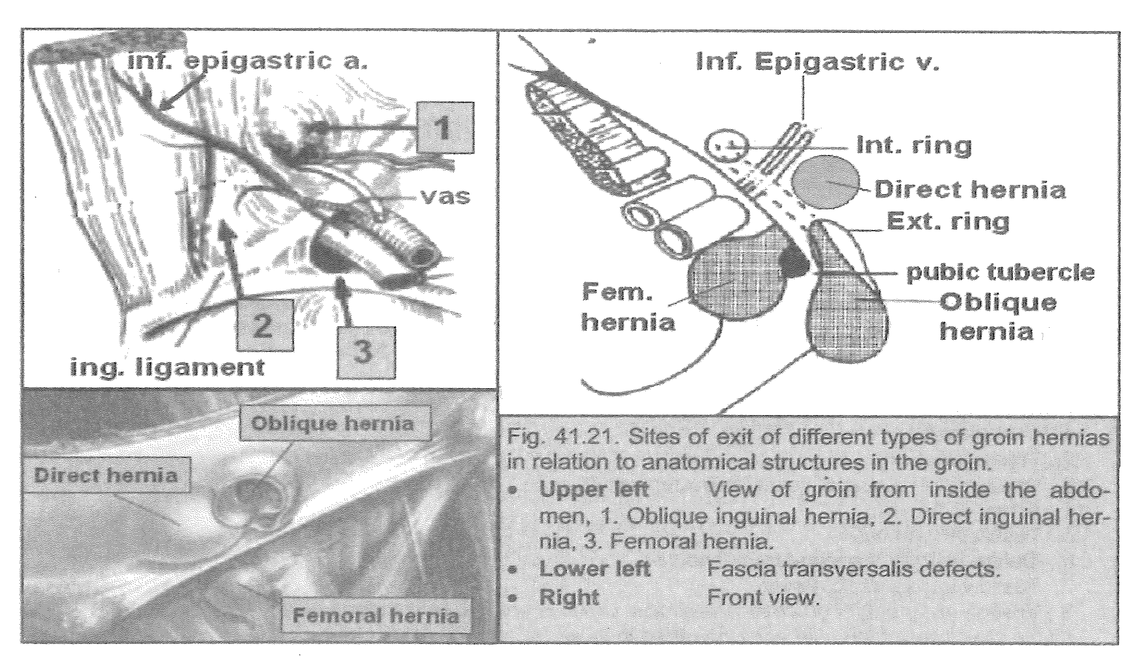


Fig. 41.21. Sites of exit of different types of groin hernias in relation to anatomical structures in the groin.

- **Upper left** View of groin from inside the abdomen, 1. Oblique inguinal hernia, 2. Direct inguinal hernia, 3. Femoral hernia.
- **Lower left** Fascia transversalis defects.
- **Right** Front view.

Oblique (indirect) inguinal hernia

Aetiology

The sac of an oblique inguinal hernia is either:

1. Congenital (preformed) sac due to the presence of an unobliterated processus vaginalis (Fig. 41.7).

2. Acquired (pulsion) sac due to raised intra-abdominal pressure and weak abdominal wall.

Anatomy of oblique inguinal hernia

1. The hernia defect is the stretched deep ring.
2. The hernia sac (congenital or acquired) escapes from deep ring and lies always inside the cord within the coverings, being anterolateral to the vas and vessels.
3. The contents are usually small intestine, omentum or both.
4. Coverings.
   a. In the inguinal region the coverings include the skin, superficial fascia, external oblique aponeurosis, then the two cord coverings in this region; cremasteric muscle and fascia and internal sperrnatic fascia.
   b. In the scrotum the coverings include the skin, non fatty superficial fascia containing the dartos muscle then the 3 cord coverings; external spermatic fascia, cremasteric muscle and fascia and internal spermatic fascia.

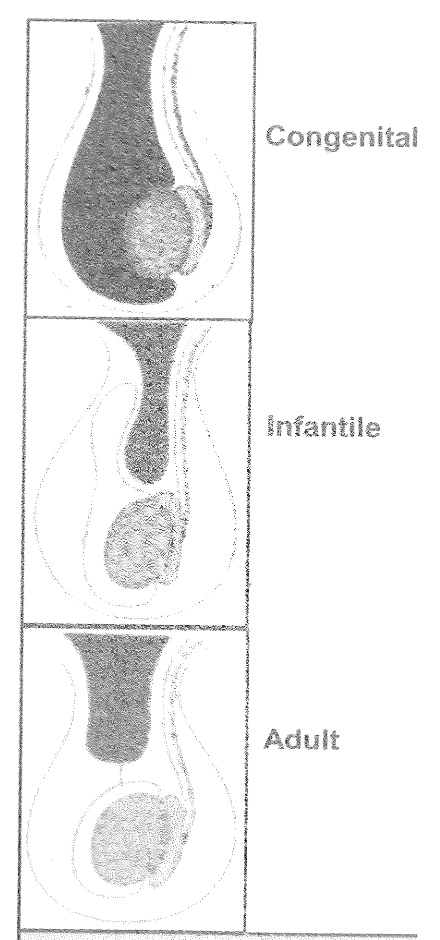
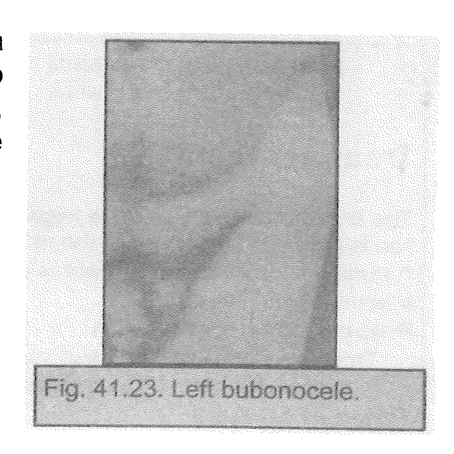


Fig. 41.22. Anatomical types of oblique inguinal hernia.

Anatomical types (Fig. 41.22)

1. **Congenital** type
   a. It is due to persistence of the whole processus vaginalis. The hernia reaches down to the bottom of the scrotum (scrotal or complete hernia).
   b. The testis lies among the contents of the sac, i.e., the testis lies within the lower part of the hernia.
   c. Although called congenital, it may appear in adult life.
2. **Infantile** type (operative finding). The tunica vaginalis extends upwards to the external ring, so that the sac passes down behind it. At operation, the tunica is liable to be opened in mistake for the true sac which will be found behind it (two sacs).
3. **Adult** type, which may be:
   a. Bubonocele
      i. The hernia is limited to the inguinal canal and is seen as a bulge or a swelling in the groin.
      ii. The processus vaginalis is obliterated at the superficial inguinal ring.
   b. Funicular hernia. The hernia reaches down to the neck of the scrotum.
      i. The processus vaginalis is closed only at its lower end, just above the epididymis.


Fig. 41.23. Left bubonocele.

ii. The tunica vaginalis is normal and the sac represents the proximal part of processus vaginalis only.
iii. The testis can be felt separate from the hernia and below it.

c. Complete (scrotal) hernia
    i. The hernia descends to the bottom of the scrotum.
    ii. The testis is behind the hernia and is difficult to locate.

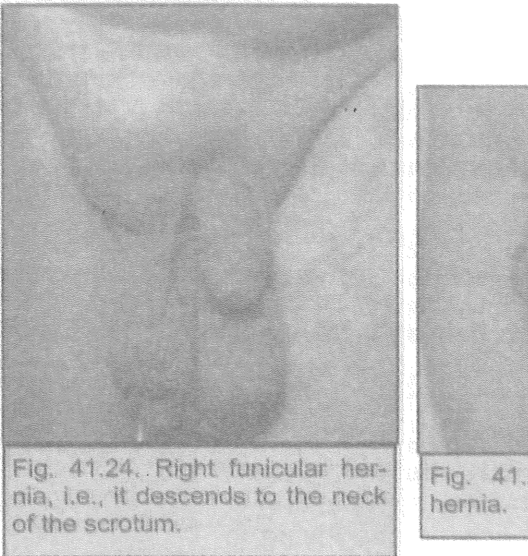


Fig. 41.24. Right funicular hernia, i.e., it descends to the neck of the scrotum.

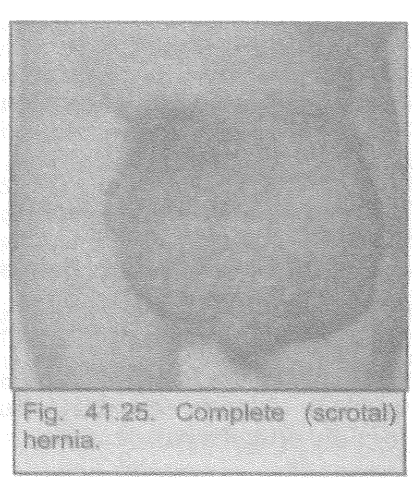


Fig. 41.25. Complete (scrotal) hernia.

Clinical features

1. Painless inguinal or inguino-scrotal swelling. Sometimes there is mild groin pain in early stages. The presence of severe acute pain indicates a complication.
2. Swelling shows an expansile impulse on cough and is reducible.
3. Site. It may be limited to the inguinal canal forming an inguinal swelling (bubonocele) or it may extend to the scrotum forming an inguino-scrotal swelling (funicular and scrotal types). Inguinal hernias lie above the inguinal ligament, and above and medial to the pubic tubercle. In contrast femoral hernias lie below the inguinal ligament, and below and lateral to the pubic tubercle.
4. Direction of descent of contents is downwards, forwards and medially.
5. Direction of reduction is obliquely upwards, laterally and backwards.
6. Shape. The swelling is oblong with a narrow neck and wide fundus.
7. Internal ring test to differentiate oblique from direct hernia. This test is not needed if the hernia is scrotal as in such case it is sure to be of the oblique variety.
    a. The patient lies down and flexes the knees to relax the abdominal muscles.
    b. The hernia is reduced by grasping it by one hand, and squeezing it upwards and laterally, while other hand manipulates at the external ring to push the contents backwards.
    c. The deep ring is determined. It lies half an inch above the mid inguinal point.
    d. The deep ring is pressed by the thumb and the patient stands up and coughs; an oblique hernia does not come out except after release of the thumb while a direct hernia comes out despite occluding the internal ring, as it comes directly from posterior wall of inguinal canal.

Treatment

The treatment of an oblique inguinal hernia is by surgery. Operation is necessary because of the risk of strangulation.

A truss is indicated only if there is a contraindication to surgery.

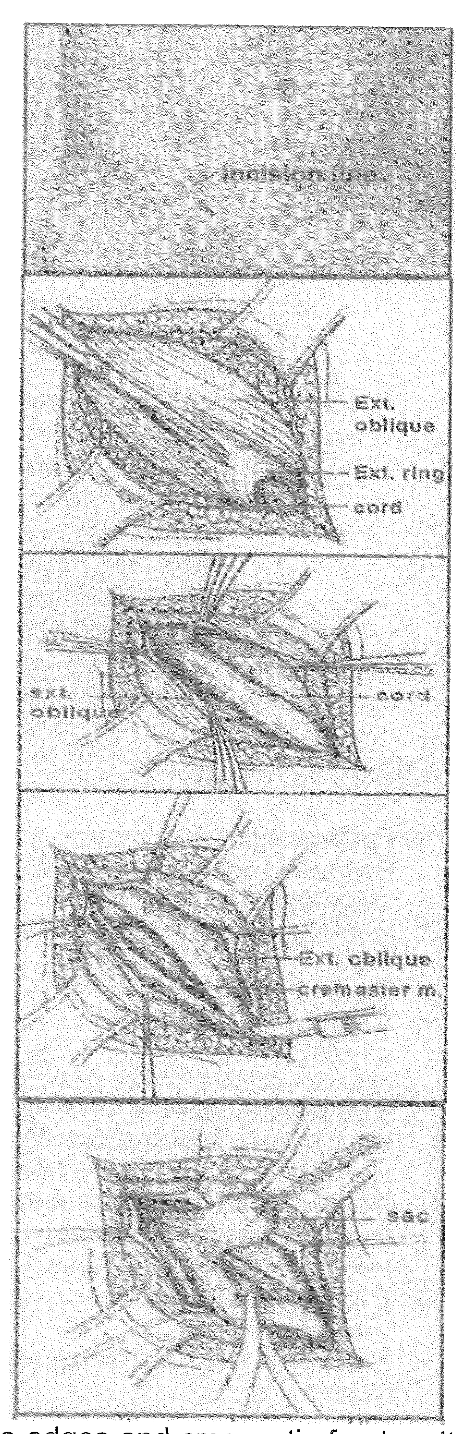
Preoperative preparation

Treat any cause of increased intra-abdominal pressure as difficulty in micturition, bronchitis, ascites, abdominal swellings, or obesity.

Types of surgery

A. Herniotomy

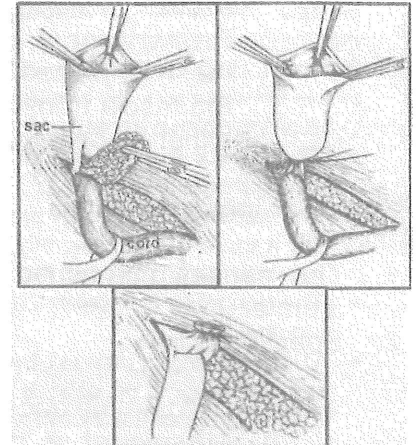
This means excision of the sac only and is performed in cases of hernias in infants and children.



Technique (Fig. 41.26- continued next page)

1. Anaesthesia. General, spinal or local anaesthesia.
2. An inguinal incision is made half an inch above and parallel to medial two thirds of the inguinal ligament. The two layers of the superficial fascia are incised.
3. The external oblique aponeurosis is incised in the direction of its fibres and the superficial ring is slit open. The spermatic cord is seen lying in the canal with the conjoint tendon arching over it. The ilioinguinal nerve traverses the canal in front of the cord and comes out through the superficial ring. In infants, the inguinal canal is short and the superficial ring lies opposite the deep ring. There is no need to open the external oblique because the surgeon can easily reach the neck of the sac without opening the inguinal canal.
4. The upper flap of the external oblique aponeurosis is dissected up to expose the conjoint tendon. The lower flap is dissected down to expose the infolded surface of inguinal ligament.
5. The cord or the round ligament is elevated to clear the posterior wall of the canal (transversalis fascia and the conjoint tendon medially).
6. The coverings of the cord (cremasteric muscle and fascia then the internal spermatic fascia) are opened longitudinally. The hernia sac is identified by its opaque pearly white colour, definite edges and crescentic fundus. It lies anterolateral to the vas and vessels. The sac is dissected up to its neck which is identified by the presence of the inferior epigastric vessels at its medial side, by being the narrowest part of sac and by the presence of extraperitoneal fat.
7. The sac is opened at its fundus and explored. Adherent intestine is separated and returned to the abdomen. Adherent omentum is excised. A finger is passed in the sac to explore for the presence of a direct or femoral hernia.

8. A transfixation ligature is applied to the neck. The sac is divided leaving a half inch stump distal to the ligature to avoid its slipping. The stump retracts up to lie flush with peritoneum.
9. The cord is returned back in place and its coverings are stitched.
10. The external oblique aponeurosis is closed. Medially, it is sutured comfortably around cord thus narrowing external ring if wide.
11. Skin closure by silk.



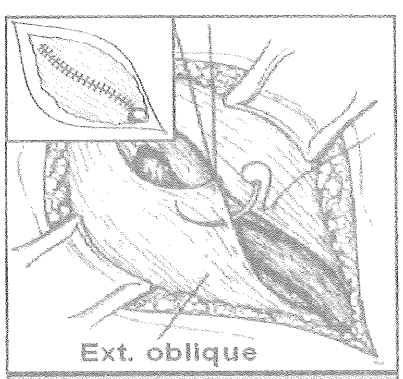


Fig. 41.26. Steps of inguinal herniotomy.

B. Herniorrhaphy

- Herniorrhaphy is indicated for hernias of adults.
- The idea is to strengthen the posterior wall of the inguinal canal.
- Excision of the sac (herniotomy) should be done first.
- As a rule, repair of any hernia defect (hemiorrhaphy) should be performed by nonabsorbable suture material as polypropylene (prolene).
- In all patients plication of the fascia transversalis and reconstruction of the internal ring with lateral displacement of the cord are performed. The placating sutures pass through the arch of transversus abdominis superiorly and the iliopubic tract inferiorly (Fig. 41.27). Then an additional procedure is added, and in any of them the repair should not be under tension.
    - **Bassini's repair**. The inguinal ligament is sutured to the aponeurotic part of the conjoint tendon behind the spermatic cord (Fig. 41.28). To be successful, the gap between the two structures should not be wide and the conjoint tendon should be strong.
    - This operation is performed for young patients.
    - Previous operations as shouldice or McVay's repair are not commonly performed now.

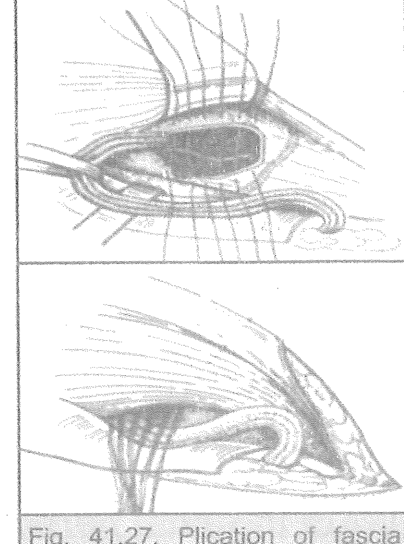


Fig. 41.27. Plication of fascia transversalis and reconstruction of internal ring.

C. Hernioplasty

- This means obliteration of the hernia defect using tissues which are not from the vicinity of the hernia.
- It is generally indicated whenever the defect is very wide (any repair under tension is doomed to failure) or when the musculoaponeurotic boundaries are too weak to hold sutures. Currently hernioplasty is gaining popularity for the treatment of all adult inguinal hernias because it is not associated with tissue tension and consequently has the lowest recurrence rate.
    - o **Onlay mesh hernioplasty**. After excision of the sac a mesh which is made of synthetic material is placed behind the spermatic cord (Fig. 41.29), and is fixed to the transversus abdominis and its aponeurotic

arch superiorly, and to the iliopubic tract and the inguinal ligament inferic The interstices of the mesh will be impregnated with a dense sheet of fibr tissue. This operation is called **Lichtenstein tension free** mesh repair.

- **Preperitoneal hernioplasty**. In this case the mesh is placed between the peritoneum and the fascia transversalis. This can be achieved either by open or by laparoscopic surgery.

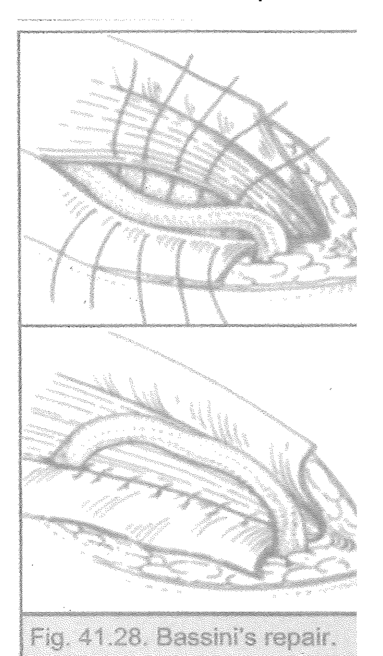


Fig. 41.28. Bassini's repair.

Recurrent inguinal hernia

Aetiology

1. The causes mentioned before leading to incisional hernia.
2. Specific causes.
   a. Leaving a part of the original sac, i.e., failure to ligate the sac at the proper neck.
   b. Missing of a direct hernia sac which was present in addition to the oblique one.
   c. Failure to do the proper repair, e.g., doing a Bassini's repair in a patient with a weak conjoint tendon or doing the repair under tension.

In most of the patients with a recurrence after a repair of an oblique inguinal hernia, the recurrence will be in the medial end of the repair and will present as a direct inguinal hernia.



Treatment

- Correction of any predisposing factors. A truss may be applied until the patient is fit for surgery.
- Hernioplasty by a synthetic mesh is usually performed.

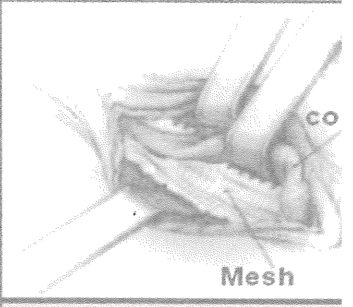


Fig. 41.29. Onlay mesh hern plasty.

Inguinal hernia in infants and children

- The hernia is always due to the presence of a preformed sac.
- It is always of the oblique type (Fig. 41.31).
- Operation can be performed at any age provided a skilled anaesthetist is available.
- Herniotomy alone is performed. In infants the operation can be performed through the external ring without the need to open the inguinal canal.
- Recurrence is rare and is due to failure to ligate the sac at the proper neck.
- If strangulation is neglected, testicular atrophy may occur.

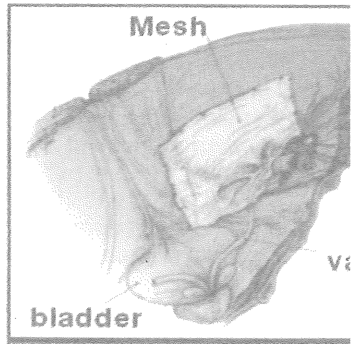


Fig. 41.30. Preperitoneal m hernioplasty.

Direct inguinal hernia

Aetiology

Direct inguinal hernia is common in elderly males. Often the patient has weakness of the lower abdominal

muscles with chronic cough or straining due to urinary problems. Injury of the ilioinguinal nerve during appendicectomy causes paralysis .of conjoint tendon which may lead to directinguinal hernia.

Pathology

Direct inguinal hernia protrudes through Hasselbach's triangle which is bounded medially by lateral border of rectus muscle, laterally by the inferior epigastric vessels and inferiorly by the medial half of the inguinal ligament (Fig. 41.21).

Clinical features

- Direct inguinal hernia usually affects old males.
- Bilaterality is common (Fig. 41.32).
- Complications are unusual.

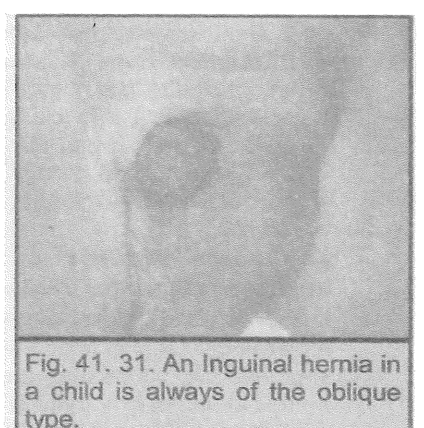


Fig. 41. 31. An Inguinal hernia in a child is always of the oblique type.

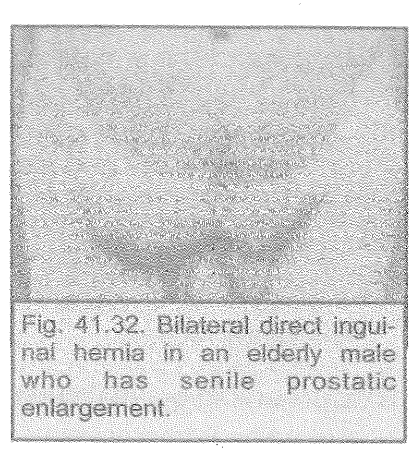


Fig. 41.32. Bilateral direct inguinal hernia in an elderly male who has senile prostatic enlargement.

The differences between the direct and indirect inguinal hernia are summarized in table 41.3.

Table 41.3. Differences between oblique and direct inguinal hernia

	Oblique	**Direct**
Age	Any age	Elderly
Side	Uni or bilateral	Commonly bilateral
Shape	Oblong	Hemispherical
Direction of descent	Downwards, forwards and medially	Forwards
Direction of reduce tion	Upwarda, backwards and laterally	Backwards
Descent to scrotum	May occur	Very rare
Size	May attain a large size	Usually small
Complications	Liable to occur	Rare
Int. ring test	Hernia does not protrude	Hernia protrudes
Relation of neck of sac to inferiorepigastric artery at operation	Lateral to the artery (Fig. 41.21)	Medial to the artery (Fig. 41.21)

Treatment

Treatment is essentially surgical.

1. Removal of the cause of straining. In an elderly male with senile enlargement of the prostate, prostatectomy should precede hernia repair.
2. Operation. Herniotomy is not usually needed as the sac is small and is composed mainly of extraperitoneal fat. Repair of the weak posterior wall of the inguinal canal by plication of the fascia transversalis and mesh hernioplasty is indicated.

A truss may be indicated if the patient is not fit for surgery.

Femoral hernia

Surgical anatomy of the femoral canal

- The femoral canal is the most medial compartment of the femoral sheath. The intermediate compartment is occupied by the femoral vein and the lateral one by the femoral artery (Fig. 41.34).
- The femoral canal is about 1/2 inch long.
- The femoral canal is cone shaped, its mouth (femoral ring) is open upwards behind the inguinal ligament and its apex is below and is formed by fusion of the medial border of femoral sheath and the septum between the femoral canal and the femoral vein.
- Contents of femoral canal are fat, lymphatics and one lymph node of Cloquet.
- Its function is to give a space for expansion of the femoral vein.
- Relations of the femoral ring are (Fig. 41.35):
    - Anteriorly: Inguinal ligament (Poupart's ligament).
    - Posteriorly: Pectineal fascia and pectineal ligament (Cooper's ligament).
    - Laterally: Femoral vein.
    - Medially: Lacunar (Gimbernat's) ligament.

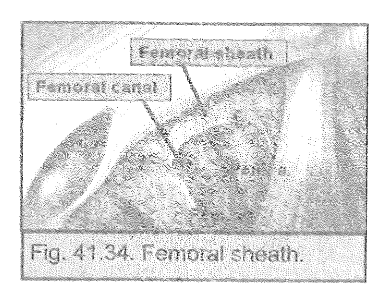


Fig. 41.34. Femoral sheath.

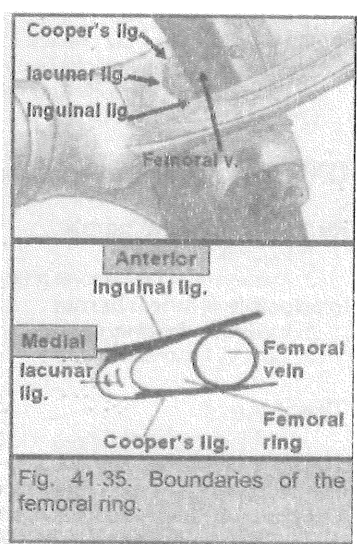


Fig. 41.35. Boundaries of the femoral ring.

Femoral hernia components

Sac. The sac of a femoral hernia proceeds downwards in the femoral canal (Fig. 41.36) then forwards stretching the cribriform fascia then upwards and laterally towards inguinal ligament (Fig. 41.37). The neck of the sac is narrow, therefore femoral hernia is liable to irreducibility and strangulation which are common.

Contents. A femoral hernia usually contains omentum, bowel or only part of the circumference of bowel (Richter's hernia).

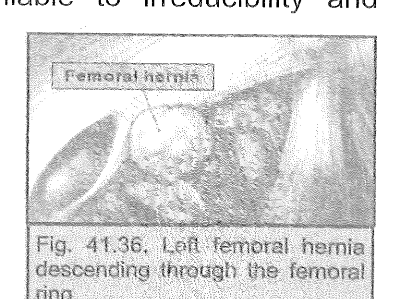


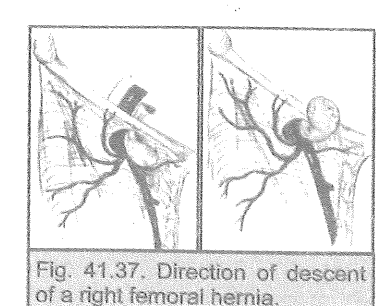
Fig. 41.36. Left femoral hernia descending through the femoral ring.

Coverings

1. Stretched femoral septum.
2. Transversalis fascia from the anterior wall of the canal.
3. Cribriform fascia.
4. Superficial fascia.
5. Skin.

Clinical features

- Femoral hernia is more common in females especially after repeated pregnancies.
- The hernia presents as a rounded swelling, below the medial part of inguinal ligament, and below and lateral to the pubic tubercle (Fig. 41.38).
- It gives an expansile impulse on cough.


Fig. 41.37. Direction of descent of a right femoral hernia.

- The direction of reduction is downwards then backwards and finally upwards.
- Pressure on the saphenous opening obliterates the impulse and prevents descent of the hernia, but pressure on the internal inguinal ring fails to do so.
- A femoral hernia may present for the first time with strangulation. There is an acutely painful groin swelling and sometimes features of intestinal obstruction. The hernia is tense, tender, irreducible with no impulse on cough.

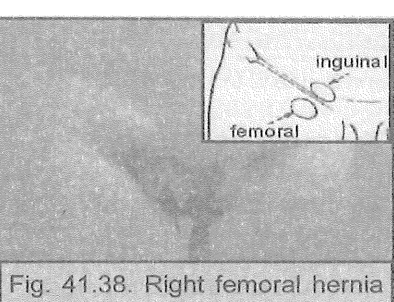
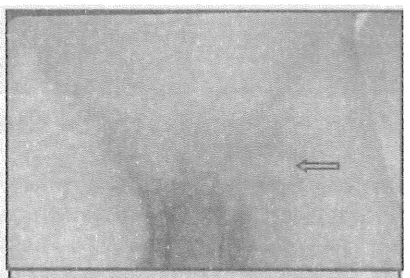


Fig. 41.38. Right femoral hernia in a female.
A femoral hernia exits below and lateral to the pubic tubercle.
An inguinal hernia exits above the pubic tubercle.


Fig. 41.39. This swelling can be any of those mentioned under differential diagnosis except for an inguinal hernia because it lies below and lateral to the pubic tubercle.

Differential diagnosis (Fig. 41.39)

Reducible femoral hernia

1. Inguinal hernia.
2. Saphena varix.
3. Aneurysm of the femoral artery.
4. Psoas abscess.

Irreducible femoral hernia

1. Irreducible inguinal hernia.
2. Lipoma.
3. Inguinal lymphadenopathy.
4. Iliopsoas bursa.

Treatment

A truss is contraindicated because the possibility of strangulation is inguinal hernia because it lies high. **Surgery is the only line of treatment,**

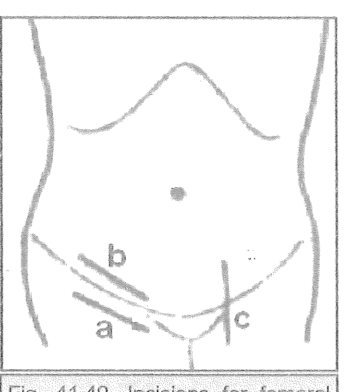
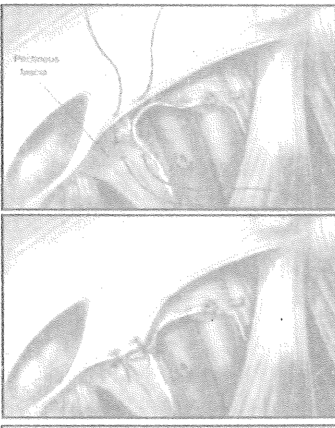


Fig. 41.40. Incisions for femoral hernia repair.
- a for the low approach
- b for the high approach
- c for preperitoneal approach (McEvedy's operation)


Fig. 41.41. Low approach to femoral hernia repair. The inguinal ligament is sutured to Cooper's ligament with non-absorbable sutures.

Approach. There are three approaches for the repair (Fig. 41.40).

- **The low approach**. The incision is 1/2 inch below and parallel to the inguinal ligament (Fig. 41.41). The sac is transfixed as high as possible and the femoral ring is closed by suturing the inguinal to Cooper's ligament.

- **The high approach** (Lotheissens operation). An incision is made above and parallel to the medial 2/3 of the inguinal ligament (similar to that of inguinal hernia). The external oblique aponeurosis is incised so that the inguinal canal is opened and the cord is isolated upwards. Transversalis fascia in the floor of inguinal canal is incised

medial to the inferior epigastric vessels exposing the peritoneum and the neck of the sac as it enters the femoral canal.

- **The preperitoneal approach** is suitable for both the uncomplicated and the complicted femoral hernias. It is either done through a lower midline incision or a pararectal incision (McEvedy's operation) at the outer border of the lower part of the rectus abdominis. The latter is extended down to beyond the inguinal ligament in case of strangulation to empty the sac of its toxic contents before releasing the strangulation agent. The abdominal incision is deepened dividing the fascia transversalis till the peritoneum and the protruding sac are exposed.
- The sac is excised.
- The defect (femoral ring) is obliterated by either:
  - Suturing the inguinal ligament (anterior border of femoral ring) to Coopers ligament (posterior border of femoral ring) by interrupted polypropylene sutures.
  - Synthetic mesh as for inguinal hernia (Fig. 41.30).

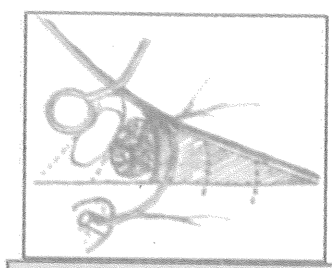


Fig. 41.42. Abnormal obturator artery passes over the lacunar ligament.

Strangulated femoral hernia

Strangulation is common because the neck of the sac is narrow and the constricting agent which is the crescentic edge of Gimbernat's ligament is sharp.

Intestinal obstruction is absent if the content of the sac is omentum, Meckel's diverticulum (littré's hernia) or part of the circumference of bowel (Richter's hernia).

Treatment

- Urgent surgery, preferably by the McEvedy's operation (Fig. 41.40c), is indicated.
- The sac is exposed, and the fundus is opened to evacuate the toxic fluid. The femoral ring is then exposed from above and the lacunar ligament is incised against the finger within the neck of sac. In dealing with the lacunar ligament, avoid injury of the abnormal obturator artery which is present in 30% of cases (Fig. 41.42).
- The contents are delivered above the inguinal ligament and are dealt with as usual.
- During repair of a femoral hernia the surgeon should be aware of the femoral vein on the lateral side and of the urinary bladder on the medial side.

Umbilical hernia

There are 3 types of umbilical hernia:

1. Congenital umbilical hernia.
2. Infantile umbilical hernia.
3. Adult umbilical hernia is usually seen in patients with ascites.

Congenital umbilical hernia (exomphalos)

The infant is borne with an umbilical hernia. There are 2 types (Fig. 41.43) of congenital umbilical hernia (exomphalos):

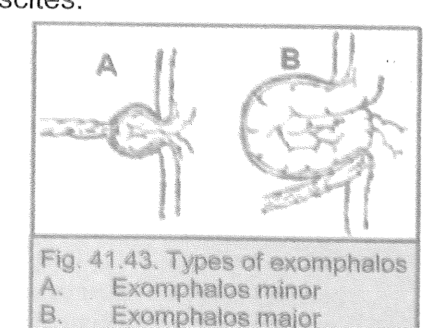


Fig. 41.43. Types of exomphalos
A. Exomphalos minor
B. Exomphalos major

Exomphalos minor

- A small defect (less than 5 cm) is present at the umbilicus through which a small peritoneal sac protrudes.
- The contents are usually intestine or Meckel's diverticulum.

- The coverings are a thin layer of Wharton's jelly and a layer of amniotic membrane.
- Treatment. The contents are reduced and returned to the abdomen, the sac is excised and the defect is repaired in layers.

Exomphalos major (Fig. 41.44)

- There is a large defect (more than 5 cm) in the center of the abdominal wall, usually above the umbilical cord.
- The contents may include many viscera and occasionally a part of the liver.
- The covering is only a layer of amniotic membrane. There is a danger of rupture of the sac followed by peritonitis.
- Treatment is by urgent operation. Usually there is no room in the abdomen to accommodate the contents.
    - If the sac is intact, the defect is closed by a synthetic mesh.
    - If the sac has ruptured skin flaps are used. The skin on either sides of the defect is undermined creating skin flaps which are brought together over the sac and sutured. Release incisions in the flanks are needed. After several months the peritoneum and muscles can be approximated and closed in layers.

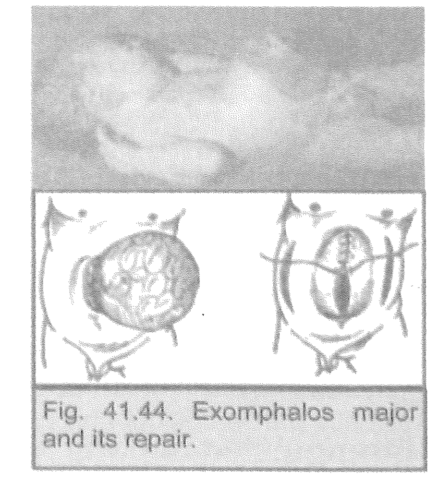


Fig. 41.44. Exomphalos major and its repair.

Infantile umbilical hernia

Aetiology

This hernia may be due to:

- Weakness of the umbilical scar from infection of the umbilical cord stump
- Increased intra-abdominal pressure from coughing or abdominal distension.

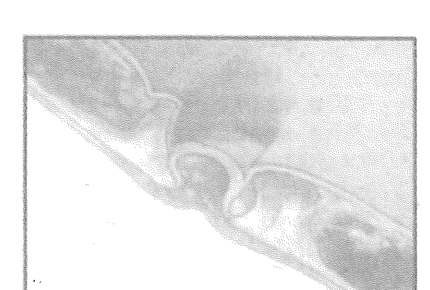


Fig. 41.45. In the infantile type of umbilical hernia the defect is exactly at the umbilicus.

Pathology (Fig. 41.45)

- The defect is exactly at the umbilicus.
- The umbilical scar is stretched and is present at the top of the hernia.
- The neck of the sac is wide and the coverings are extraperitoneal fat and umbilical scar.

Clinical features

- There is umbilical protrusion which increases with cough or crying (Fig. 41.46).
- The edge of the defect can be palpated as a firm ring.
- Obstruction and strangulation are rare below the age of 3 years.
- This type occasionally affects adults.

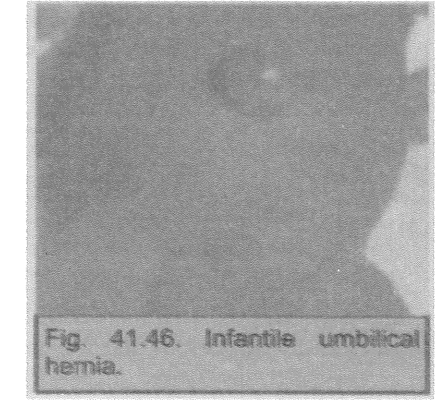


Fig. 41.46. Infantile umbilical hernia.

Treatment

Reassurance of the parents and follow-up are the usual measures. The defect closes spontaneously within two years in most of the cases.

- Correction of the cause of straining, if any.
- Operation is indicated when the defect is more than 2 fingers wide or when the hernia persists after the age of two years. A semicircular incision is done below the umbilicus, and a skin flap is turned upwards. The sac is transfixed and excised, and the defect in linea alba is closed with few stitches of non-absorbable suture material as polypropylene (prolene).

Adult umbilical hernia (the principles of the treatment are the same as paraumbilical hernia)

Paraumbilical hernia

Aetiology

This hernia is more frequent in middle aged females, especially in obese multiparous women. It is actually paraumbilical and not umbilical hernia.

Fig. 41.47. A small and a large paraumbilical hernias.

Surgical pathology

- Paraumbilical hernia defect lies in the linea alba. In most cases it is situated above the umbilicus because the linea alba is thinner and broader above than below the umbilicus. Occasionally the defect is below the umbilicus. It is never lateral to it.
- The umbilical scar lies below the swelling. It is compressed by the hernia and looks like a crescent.
- The sac has a narrow neck with a small defect in the linea alba. Adhesions inside the sac are very common especially at the fundus, rendering the hernia irreducible. Complications as strangulation and irreducibility are very common due to the narrow neck, sharp edge and adhesions inside the sac.

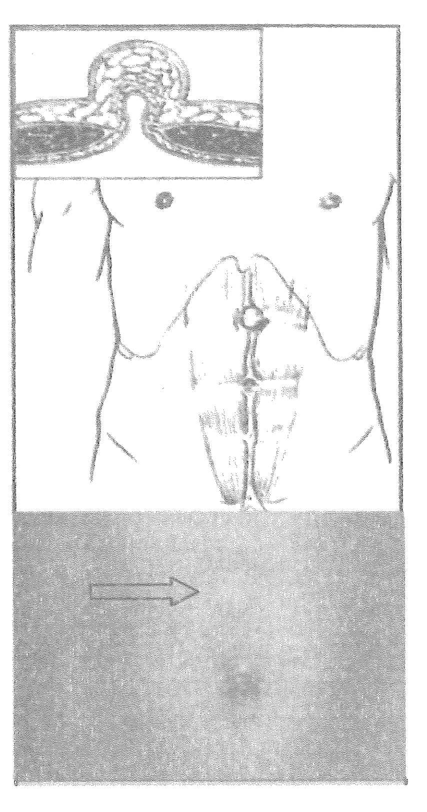
Clinical features

- There is a painless swelling above the umbilicus (Fig. 41.47) which gives an expansile impulse on cough.
- Mild dragging pain may be present in a huge hernia. Severe acute pain indicates strangulation.
- Paraumbilical hernias are frequently found to be irreducible or partially reducible. They are liable to strangulation.

Treatment

- **Surgery is the only method of treatment.** A truss is not satisfactory because the hernia is usually irreducible and its use carries a high risk of strangulation.
- For the obese reduction of weight is advised prior to surgery.
- An elliptical transverse incision is made over the maximum convexity of the hernia and skin flaps are undermined upwards and downwards. The sac is exposed and dissected down to the neck. The sac is opened at its neck-because adhesions are usually present at the fundus. The contents are dealt with and reduced into the abdomen. The sac is excised at the neck. The defect is then repaired:

- If the defect is the small it is closed by non absorbable suture.
- If the defect is large or the musculo-aponeurotic layer is weak, a prolene mesh is used to close the defect.
- The previous Mayo's repair is not commonly performed nowadays.


Fig. (41.48): Epigastric hernia

Epigastric hernia

This hernia starts as a protrusion of the extraperitoneal fat through a defect in the supraumbilical part of the linea alba and is called "fatty hernia of linea alba". As the protrusion enlarges the fat pulls through the defect a small peritoneal pouch which may contain intestine or omentum, and is called "epigastric hernia".

Clinical features

- Epigastric hernia may be symptomless, it may cause local pain or it may cause dyspepsia due to traction on the stomach.
- There is a swelling in the epigastrium which is soft, frequently irreducible and gives an expansible impulse on cough (Fig. 41.48). Occasionally there are multiple hernias.

Treatment

If there is pain, the surgeon should be sure that it is not due to an underlying disease, e.g. peptic ulcer or gallstones.

Operation is performed by excision of the protruding extraperitoneal fat and the hernia sac followed by simple closure of the linea alba defect. If the defect is large, a prolene mesh hernioplasty is performed.

Divarication of recti

This is separation of the recti due to stretching of the linea alba by a chronically raised intra:abdominal pressure.

Clinical features

- Divarication of recti is common in middle aged females due to repeated pregnancies and in patients who have ascites and splenomegaly.
- When the abdomen is relaxed, nothing is visible, but on raising the shoulders, the linea alba bulges as a longitudinal ridge and the fingers can be dipped into the abdomen between the two recti.

Treatment

An abdominal belt is satisfactory in most cases. Surgical repair is likely to fail until the cause of high intra-abdominal pressure is treated.

Incisional hernia

Incisional hernia is a hernia that develops at the site of a previous abdominal incision. The aetiology and clinical picture have been discussed before. The commonest cause is surgical site infection (Fig. 41.49).

Treatment

If the patient is unfit for surgery and provided the hernia is reducible, an abdominal binder will keep the hernia reduced.

Surgery offers the only definitive cure. Many operations are available, but it is to be stressed that any repair under tension is doomed to failure.

1. **Anatomical repair**. The idea is to expose the hernia defect, remove the sac and then repair the abdominal wall in layers according to the site of the incision. If the sac has a wide neck it is not necessary to excise. It is just pushed inside and is covered by the repair.
2. **Hernioplasty**. If the hernia defect is wide or the musculoaponeurotic edges are weak, it is recommended to perform hernioplasty using a synthetic mesh (Fig. 41.50).

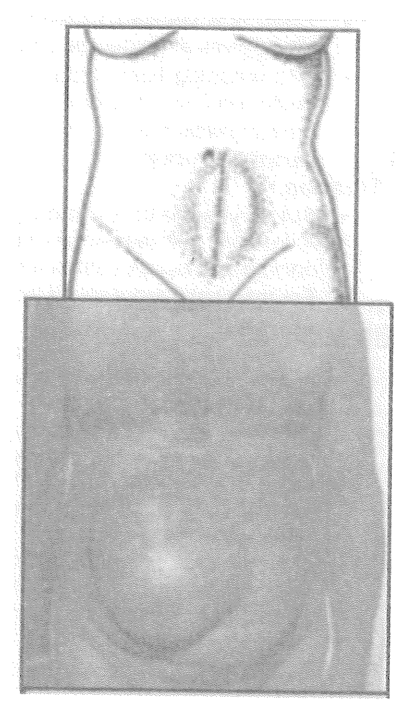


Fig. (41.49): Incisional hernia at the site of midline incision

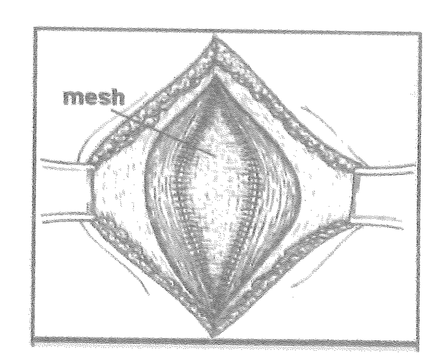


Fig. (41.50): Mesh hernioplasty for incisional hernia repair

DIAGNOSIS OF URINARY TRACT DISEASES

While most of the urinary tract (UT) is hidden deeply in the abdomen and the pelvis, a systematic approach to its diseases leads to an accurate diagnosis.

CHAPTER CONTENTS

Symptoms of urinary tract diseases

Systemic symptoms

1. Symptoms of toxemia. Fever, malaise and headache accompany pyelonephritis and pyonephrosis. The latter may be accompanied by rigors as well.
2. Symptoms of metastasis. Malaise, weight loss, back bony pain and symptoms related to involved organ.
3. Symptoms of uremia (renal failure). Pruritus, malaise, nausea, vomiting, lassitude, forgetfulness, behavior changes, loss of weight, loss of libido, headache and bleeding tendencies as epistaxis and melena.

Upper urinary tract symptoms

Renal pain (Fig. 42.1) is a dull constant aching pain in the renal angle (angle between the sacrospinalis and the last rib) that is referred anteriorly to the anterior renal point (at upper outer quadrant of the abdomen). Usually it is due to renal pelvis distension or to stretch of renal capsule.

Ureteric (renal) colic (Fig. 42.1) is colicky pain that extends from the loin to the groin. Ureteric colic is usually accompanied by nausea and vomiting. It is due to adute obstruction by a stone, which leads to distension and increased muscular contractions (i.e. peristalsis) of the ureter. The pain is localized to the loin when the stone is just coming out of the kidney. As the stone moves downwards, the pain is felt in the groin and may radiate to the upper thigh and to the scrotum in males or the vulva in females. When the stone is impacted at the lower end of the ureter, there are symptoms of vesical irritation and pain referred to the tip of the penis.

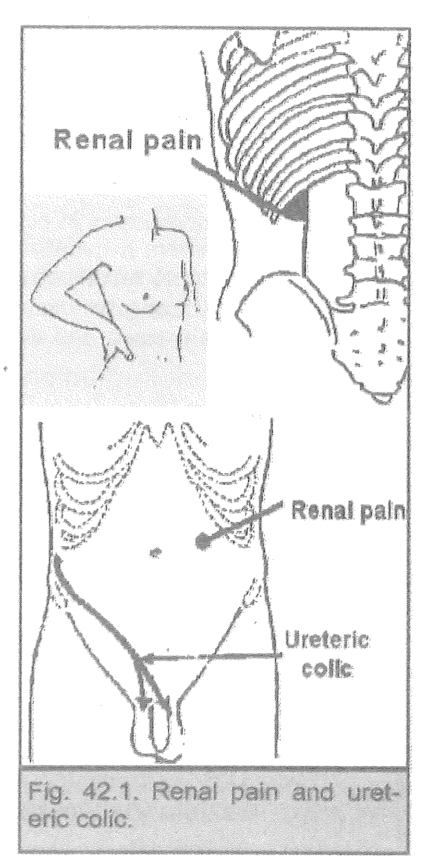


Fig. 42.1. Renal pain and ureteric colic.

Lower urinary tract symptoms

1. Pain

Vesical pain varies from slight discomfort to severe agonizing pain in the suprapubic region especially with a full bladder. It is referred to the tip of penis with severe

desire to micturate that results in expulsion of a few drops of urine. Vesical pain is caused by cystitis, stone, or bladder carcinoma.

Prostatic and seminal vesical pain is deeply seated vague discomfort in the pelvis and is referred to the perineum, rectum or suprapubic region. It is usually due to inflammation.

Urethral pain is burning. It occurs during micturition and is caused by inflammation or a stone.

Testicular and epididymal pain is severe local pain that may radiate to lower abdomen or costo-vertebral angle. It is caused by epididymo-orchitis, trauma or torsion. Dull ache may be caused by varicocele.

2. Symptoms related to micturition

Symptoms of bladder irritation can be caused by infections (cystitis, prostatitis), bladder inflammation (from chemical, thermal or radiation injury); or by a foreign body like a stone. These symptoms include:

- **Frequency of micturition:** The normal bladder capacity is about 400 ml. Frequency may be by day (diurnal) or by night (nocturnal). Frequency is caused by:
  - Irritation of bladder, e.g., cystitis (diurnal and nocturnal) and bladder stones (diurnal).
  - Mechanical reduction of bladder capacity, e.g., bladder tumours or bladder fibrosis by tuberculosis, radiation or Schistosomiasis.
  - Functional reduction of bladder capacity as in patients with prostatic enlargement due to the presence of a big amount of residual urine (nocturnal frequency).
  - Polyuria Increased urine production by the kidneys due to excessive drinking or uncontrolled diabetes.
- **Urgency**: Urgency is a strong sudden desire to urinate caused by hyperactivity and irritability of the bladder. It results from obstruction, inflammation, or neuropathic bladder disease. In most circumstances, the patient is able to temporarily control urine, but loss of small amounts of urine may occur (urgency incontinence).
- **Strangury** Strangury is a persistant desire of urination and severe straining at micturition with the passage of a few drops of urine that may be blood stained.
- **Dysuria**: Painful urination is related to acute inflammation of the bladder, urethra, or prostate. Pain is described, as "burning" on urination. Dysuria is often associated with urinary frequency and urgency.
- **Symptoms of Bladder outlet obstruction**. Prostate enlargement (benign or malignant) and urethral stricture are common causes.
  - Hesitancy and straining. Delay in initiating urinary stream is the early symptom of bladder outlet obstruction. Later, hesitancy is prolonged and the patient strains to pass urine.
  - Difficulty in micturition: In benign prostatic hyperplasia, the patient has to relax at the beginning of micturition (hesitancy), while patients with a urethral stricture have to strain to pass urine. In these patients the urinary stream is weak.
  - Loss of force and reduced caliber of the stream is noted as urethral resistance increases despite the generation of increased intravesical pressure.
  - Terminal dribbling. Becomes more noticeable as obstruction progresses.
  - Interruption of urinary stream. May be abrupt with severe pain radiating down the urethra due to vesical calculus.

- Sense of incomplete emptying. Patient feels that urine is still in the bladder after urination due to residual urine.
- Retention of urine; acute and chronic (see later).

3. Urinary incontinence (see later)

4. Symptoms related to changes in urine

- **Changes in urine volume:**
    - **Polyuria**. Excessive fluids intake, diabetes mellitus, diabetes insipidus and high-output chronic renal failure.
    - Oliguria is urine volume less than 400ml124h in adults. The causes are either acute renal failure (due to shock or dehydration), or bilateral ureteral obstruction.
    - Anuria is urine volume less than 200m1/24h. The causes are:
        - Pre-renal causes, e.g., shock and dehydration.
        - Renal causes, e.g., toxins, drugs and renal diseases.
        - Post-renal causes, e.g., bilateral renal obstruction or complete obstruction in a solitary kidney. If due to stones, it is called calcular anuria. Anuria is one of the urological emergencies.
- **Changes in color and content of urine:**
    - Haematuria is passage of blood in urine (see later).
    - Pyuria. The presence of pus in urine. It may be microscopic or macroscopic.
    - Chyluria. Passage of white milky urine due to presence of lymphatic fluid or chyle. It represents a lymphatic-urinary fistula due to obstruction of renal lymphatics, which causes forniceal rupture and leakage. Urine clears on adding ether. Adding Sudan III gives an orange colouration. Filariasis, trauma, tuberculosis, and retroperitoneal tumours may cause this problem.
    - Cloudy (turbid) urine. Urine looks turbid due to the presence of excessive phosphate crystals (phosphaturia) that precipitated in alkaline urine. Adding few drops of acetic acid clears the turbidity. Infection can also cause urine to be cloudy and malodorous.
    - Necroturia. Passing pieces of necrotic tissue in urine. It is pathognomonic of bladder carcinoma.
    - Pneumaturia. Presence of gas in urine. It is strongly suggests a fistula between the urinary tract and the bowel that is caused by malignancy of colon or bladder, Crohn's disease, or by colonic diverticulitis.

5. Other symptoms:

- Urethral discharge is one of the common complaints in men and is caused by urethritis, particularly the gonococcal type. It is accompanied by burning micturition.
- Bloody ejaculation (haemospermia). Due to inflammation of the prostate or seminal vesicles.
- Visible or palpable masses
    - A visible or a palpable mass in the upper abdomen may represent renal tumour, hydronephrosis, or polycystic kidney.
    - Enlarged lymph nodes in the neck from metastasis from testis or kidney malignancies.
- Painless masses in scrotum include hydrocele, varicocele, spermatocele, chronic epididymitis, hernia, and testicular tumour.
- **Infertility**. Varicocele may reduce sperm count and motility. Mumps orchitis may cause testicular atrophy.

- **Sexual difficulties in men**. Impotence, premature ejaculation, and even loss of desire.
- Gynaecomastia is a common finding in prostate cancer patients who take oestrogen therapy. It may also be seen in patients who have oestogen-secreting testicular choriocarcinoma.

Examination for urinary tract diseases

Signs of chronic renal failure may be present, e.g. pallor from anaemia, eye puffiness, dry tongue, abnormal odor of breath, abnormal respiration, drowsiness, dehydration, abdominal distension, tremors, convulsions, oedema of lower limbs and hypertension.

Abdominal examination

Inspection, palpation, percussion and auscultation for masses or fullness in rena angle or suprapubic area, scars, tenderness and auscultation of systolic bruit over rena artery stenosis.

Differential diagnosis of a renal mass

1. Hydronephrosis.
2. Pyonephrosis
3. Polycystic kidney.
4. Renal tumours (hypernephroma or Wilm's tumour).
5. Big renal cyst.

Differential diagnosis of a suprabupic swelling

1. Full bladder due to acute or chronic retention of urine.
2. Pregnancy.
3. Bladder tumour.
4. Uterine or ovarian masses.

Genital examination for ulcers, meatal stenosis, hypospadius, epispadius discharge, swelling, hydrocele, varicocele. . .etc. Vaginal exam in females.

Differential diagnosis of a purely scrotal swelling

1. Vaginal hydrocele.
2. Encysted hydrocele of the cord.
3. Spermatocele.
4. Haematocele.
5. Pyocele.
6. Chylocele.
7. Testicular tumours.
8. Tuberculous nodules of the cord.
9. Bilharzial mass of the spermatic cord.

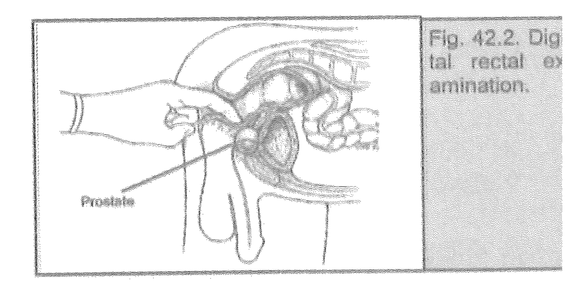


Fig. 42.2. Digital rectal examination.

Digital rectal or bimanual examination (DRE)

What to feel (Fig. 42.2)

- Anal sphincter tone.
- Walls of anal canal and rectum.
- Prostate.
  - Size (normal 4cm in length and width).
  - Nodules.

- Consistency (normal rubbery).
- Tenderness.
- Mobility.
- Seminal vesicles are normally not felt (palpable if calcified due to Bilharsiasis).
- Urinary bladder tumours are felt.

- Malignant deposits in rectovesical or recto-uterine pouch.

Differential diagnosis of a hard nodule in the prostate

1. Prostate cancer.
2. Prostatic calculi.
3. Tuberculous nodule.

Investigations for urinary tract diseases

The following is a long list of investigations. They need not be done all for every case. The choice depends on the clinical situation.

Laboratory investigations

Urine analysis (mid stream) for:

(a) Physical properties:
  a. Color; normally amber yellow.
  b. pH; normally acidic with a range 4.5-7.5.
  c. Specific gravity; normally 1005-1040 according to hydration.

(b) Biochemical analysis for albumin, glucose and bilirubin.

(c) Microscopic examination for:
  a. Crystals as oxalates, uric acid, phosphates and cystine.
  b. Casts as hyaline, granular and epithelial.
  c. Cells as RBCs or WBCs. Normal urine contains less than 5 RBCs and WBCs/HPF (high power field). Epithelial cells are present in normal female urine.
  d. Bilharzia ova and sperms.

(d) Microscopic staining for:
  a. Pyogenic organisms by Gram's stain.
  b. Acid-Fast (Ziehl-Neelsen) stain for TB.

(e) Culture of a sterile sample and antibiotic sensitivity.

(f) Cytological examinations for malignant cells.

Urethral discharge analysis and culture for gonorrhea and Chlamydia.

Renal function tests

a. Blood urea (N=15-40 mg/l 00 ml) is not an accurate test of renal function.
b. Blood urea nitrogen (BUN). (N=12-15 mg/100 ml) is more accurate.
c. Serum creatinine is the best (N=0.2-1.5 mg/100 ml).
d. Creatinine clearance test (N=70-140 ml/mm). It means the amount of plasma completely cleared from creatinine by the kidney in one minute. It denotes qualitative kidney function very accurately.
e. Serum electrolytes. Serum K is elevated in cases of renal failure.

Serum PSA (prostatic specific antigen) for the diagnosis of prostate cancer (normally 4 ng/ml).

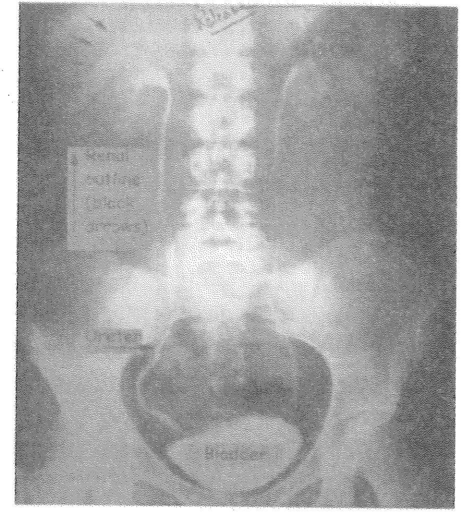
Imaging

Ultrasound

- Abdominal and pelvic ultrasound is a simple, cheep and non-invasive technique.
  - It provides information about morphology, site and size of the kidneys as well as thickness of renal parenchyma.
  - It can diagnose obstructive uropathy with dilatation of the pelvicalyceal system.
  - It can differentiate between cystic and solid lesions of the kidney.
  - Renal and bladder calculi are easily diagnosed.
  - Bladder tumours could be visualized.
  - Ultrasound can estimate the amount of post-voiding residual urine.
- **Transrectal ultrasound** is very accurate in diagnosing prostatic lesions. It allows guided biopsies from suspected prostatic cancer.

Plain X-ray of urinary tract (also known as plain UT or KUB, for kidney, ureters, and bladder). The following findings are looked for:

- Soft tissue shadow of the kidney, which is enlarged with renal swellings.
- Psoas shadow, which is obliterated by a perinephric abscess.
- Bony skeleton, Look for spina bifida, wide separation of symphysis pubis which is seen in cases of ectopia vesicae, fractures and bone metastases.
- Radio-opaque shadows
  - Urinary stones (renal, ureteric, bladder and prostatic) and gall bladder stones.
  - Abnormal calcification of urinary tract, e.g., TB renal calcifications, calcified renal tomours,
  - Bilharzial bladder (Fig. 45.9) and calcified Bilharzial seminal vesicles (honey comb appearance).
  - Calcifications outside urinary tract, e.g., adrenal calcification, mottled calcified lymph nodes (tabes mesenterica), calcified iliac vessels and pelvic phleboli.



Intravenous pyelography (IVP); also known as intravenous urography (IVU)

- The idea is to take X-ray films after IV injection of a radio-opaque (iodine containing) contrast material, e.g., urografin. The contrast is concentrated and excreted by the kidneys in urine.
- Normally a nephrogram phase appears within a few minutes and then the pelvicalyceal system is visualized. Later, the ureters and the bladder are visualized (Fig. 42.3). Afterwards the patient micturates and a post-voiding film is taken to evaluate residual urine, which is normally almost zero (Fig. 42.4).

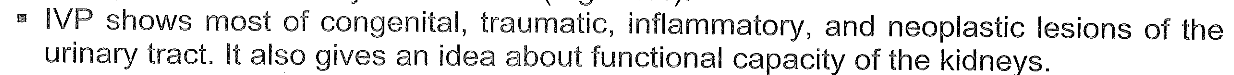


Fig. 42.3. Normal IVU.

- IVP shows most of congenital, traumatic, inflammatory, and neoplastic lesions of the urinary tract. It also gives an idea about functional capacity of the kidneys.

- Iodine contrast media are allergenic and precautions regarding sensitivity testing should be taken to avoid these reactions.

Infusion urography

- This test is indicated for patients with impaired renal function where IVU will not show up due to lack of secretion of contrast medium.
- The contrast material (urografin 2 ml/Kg) is mixed with an equal volume of saline and is infused slowly over a period of 15 minutes to give time for the kidneys to handle the contrast material. This may produce a useful urogram.
- It is done only if blood urea is less than 100mg/100 ml and contraindicated if more than this.

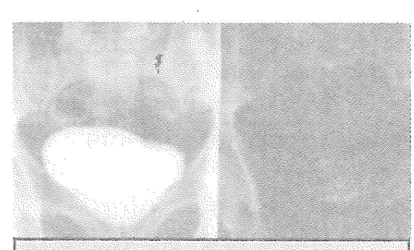


Fig. 42.4. Normal descending cystogram (left) and post-voiding film (right). Normally there should be no residual urine after voiding.

Retrograde (ascending) pyelography

- Retrograde prelography is indicated when the data obtained from IVU are not conclusive or if IVU is contraindicated (e.g. uremia or contrast sensitivity).
- A cystoscope is introduced and fine ureteric catheters are passed along the ureters to the renal pelvis and a contrast material is injected directly into the catheter.
- The test provides good delineation of the pelvicalyceal system but no information about the renal function (Fig. 42.5).

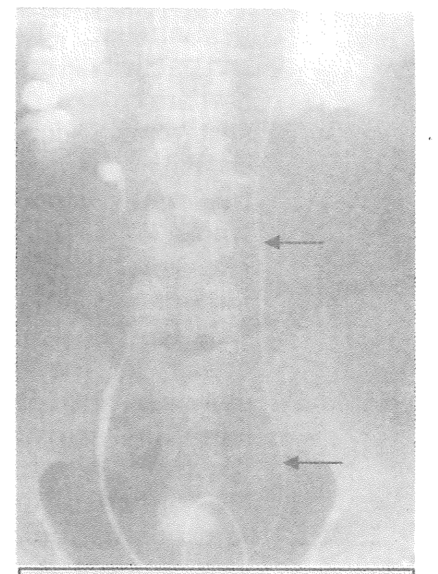


Fig. 42.5. Retrograde pyelography showing bilateral hydronephrosis. The arrows point at the ureteric catheter.

Cystography

There are two types of cystography.

- During the performance of an IVU, the bladder will be visualized in late films **(descending cystography)** (Fig. 42.4), but the pictures may not be of a high quality as the contrast is diluted with urine.
- For better visualization, a urethral catheter is passed to the bladder and 400m1 of saline mixed with contrast is infused directly in the bladder (ascending cystography) (Fig. 42.6). It is indicated to delineate bladder pathology or to diagnose vesico-ureteric reflux by taking films during voiding (voiding cystography, Fig. 45.2).



Urethrography

There are two types of urethrography.

- **Micturating urethrography** during VU.
- **Ascending urethrography** by injecting contrast medium through external meatus.

Urethrography is indicated to diagnose urethral injuries, strictures or fistulous tracts.

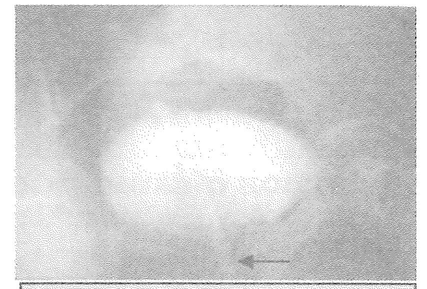


Fig. 42.6. Normal ascending cystography. The arrow points at the urethral catheter.

Renal arteriography

- Arteriography is Indicated for the diagnosis of renal artery stenosis and renal tumours, and for kidney donors before transplantation.

- Renal arteriography can be obtained by selective catheterization of the renal artery by a cannula through the femoral artery using the Seldinger technique.

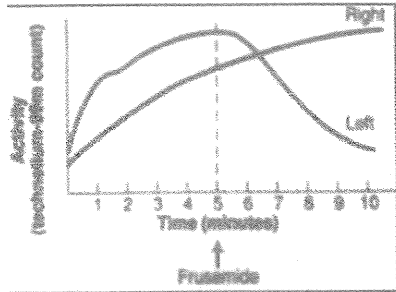


Fig. 42.7. DTPA. The left kidney shows the typical triphasic curve. The right kidney shows accentuation of isotope activity in the excretory phase, which signifies obstructed urine flow.

CT scan and MRI

Their main value is in the diagnosis and staging of genito-urinary neoplasms.

Radioisotope scanning

There are two types of renal isotope material. Both are attached (labeled) with technetium^{99m} (^{99m}Tc) and both are given intravenously.

- DTPA (diethylenetetramine penta-acetic acid), Fig. 42.7
    - The isotope is rapidly excreted through the glomerulus. After its administration, the radioactivity is traced by the use of a gamma camera.
    - A triphasic curve is plotted describing a vascular phase in which the isotope reaches the renal vasculature, a secretory phase where it is filtered by the kidney, and an excretory phase where it leaves the kidney through the ureters.
    - This type of scan is called renogram and is useful in the diagnosis of renal artery stenosis and obstructive uropathy. In the latter, the administration of furosemide, after the isotope, accentuates the abnormality in the third phase of the curve.



Fig. 42.8. DMSA isotope renography shows defective uptake in the upper and lower poles of this kidney (arrows) that denotes renal scarring.



- **DMSA (dimercaptosuccinic acid),** Fig. 42.8
    - The isotope is concentrated in the renal tubules. As only 5% is excreted, static images of the kidneys can be obtained 3 hours after injecting of the isotope.
    - DMSA is most useful in the demonstration of renal scarring that results from chronic pyelonephritis. Focal defects as tumours and haematomas, lacerations, and ischaemia can also be visualized.

Endoscopy (Fig. 42.9)

Advances in fibreoptic endoscopes have made it possible to visualize every part of the urinary tract with great accuracy.

- Endoscopy is used for both diagnostic (urethroscopy, cystoscopy, ureteroscopy and nephroscopy) and therapeutic purposes.
- Any suspicious lesion is biopsied.
- It is essential to do cystoscopy for patients with haematuria.

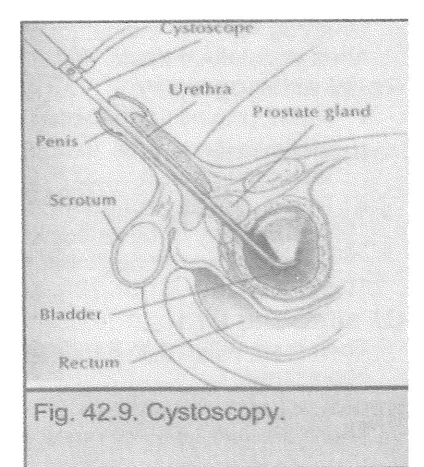


Fig. 42.9. Cystoscopy.

Urodynamic studies

These tests are used to study the physiologic functions of the bladder, urinary sphincters and urethra. They are very useful in-patients with neurogenic bladder and incontinence. These tests include:

- **Cystometry**. It measures bladder volume against intravesical pressure.
- **Flowmetry**. This is a measure of the amount of urine passed per time unit. The normal urine flow rate should be more than 20 ml/second, provided a good amount of urine is voided (not less than 120 ml). It is the most important, non-invasive and useful urodynamic test. It is frequently and commonly used to diagnose lower urinary tract obstruction e.g. senile prostatic enlargement, urethral stricture and neurogenic bladder.

Some important problems

Retention of urine

Definition

Inability to pass urine in spite of a full bladder.

Aetiology

- **Mechanical causes**
  - Urethral causes: rupture, phimosis, urethritis, stricture and stone.
  - Prostatic causes: prostatitis, abscess, senile enlargement and cancer.
  - Bladder causes: bladder neck obstruction, stone and cancer.
  - Compression from outside by a pregnant uterus, pelvic tumours or by faecal impaction.
- **Functional (neurogenic) causes**
  - Spasmodic retention as in post-operative cases, particularly after anal surgery (acute retention) and hysterical cases.
  - Atonic retention as in diabetic autonomic neuropathy and cauda equina lesions.

  Senile prostatic enlargement and postoperative retention are the commonest causes in males. Retention is much less frequent in females

Types

- **Acute retention** is sudden painful inability to pass urine. The patient has a strong desire with severe pain and tender full bladder.
- **Chronic retention**. Patient passes some amount of urine, but residual urine remains in the bladder after each act (Fig. 42.10), gradually accumulates giving a full bladder. This causes little discomfort with hesitancy and reduction of force of stream.
- **Chronic retention with overflow**. The bladder is continuously full with a large amount of residual urine and the patient passes urine only when the intravesical pressure exceeds the intraurethral pressure. Constant dribbling of urine (paradoxical incontinence) may be present. While this type is painless and the bladder is not tender, it is dangerous as it leads to back pressure changes of the upper urinary tract.

Differential diagnosis

Anuria is absence of urine or urine volume of less than 200cc/24h. The bladder is empty on contrary to retention where the bladder is full and forms a median pyriform swelling in the suprapubic area which is dull on percussion.

Treatment

Acute retention. The following measures are done in sequence.

1. Conservative treatment is tried first. An analgesic is administered. The patient is advised to get out of bed and a warm bath is advised. If there is no organic obstruction (e.g., postoperative retention), an injection of a parasympathomimetic drug as carbacol or doryl is given.
2. Catheterization is attempted if conservative treatment fails. A soft catheter is used (Nelaton or Foley). It may be removed or left as indwelling till treatment of cause.
3. Percutaneous suprapubic catheter (cystocath). If urethral catheterization fails, the urinary bladder is punctured through the abdominal wall in the suprapubic region under local anesthesia. A catheter is then introduced into the bladder.
4. Treatment of the cause, e.g., prostatectomy.

Chronic retention

1. Treatment of the cause. If there are no backpressure effects on the kidneys and renal functions are normal, there is no need for catheterization.
2. Catheterization. Self retaining Foley catheter should be inserted if there is backpressure on kidneys or elevated renal functions. Evacuation of the bladder in these cases should be very slowly to avoid bleeding from either prostatic veins or renal decompression bleeding. The cause of chronic retention is investigated and treated later.

Urinary incontinence

Definition

Uncontrolled escape of urine.

Types

1. True incontinence is urine loss without warning secondary to defective urethral sphincters, e.g., ectopia vesicae, epispadias, ectopic ureteral orifice and sphincteric injury after prostatectomy.
2. Stress incontinence is urine loss in association with physical strain, e.g., coughing, laughing and straining. It is common in multiparous women with weak muscle support of the bladder neck and urethra.
3. Urge incontinence is involuntary loss of urine with strong desire of micturition. It occurs with acute cystitis and in cases of upper motor neuron lesions.
4. Overflow (false) incontinence' is loss of urine due to chronic urinary retention or secondary to a flaccid bladder. The intravesical pressure finally equals the urethral resistance; urine then constantly dribbles.
5. Enuresis is bedwetting at night. It is physiologic during first 2-3 years of life. It may be functional secondary to delayed neuromuscular maturation of urethrovesical component, but it may be a symptom of organic disease (e.g. neurogenic bladder).
6. Fistula is an abnormal communication between urinary and gynecological organs like vesico-vaginal and uretro-vaginal fistula that develops from obstetric trauma.

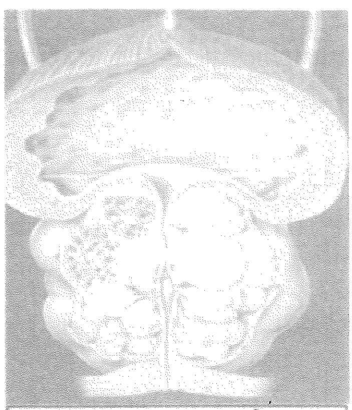
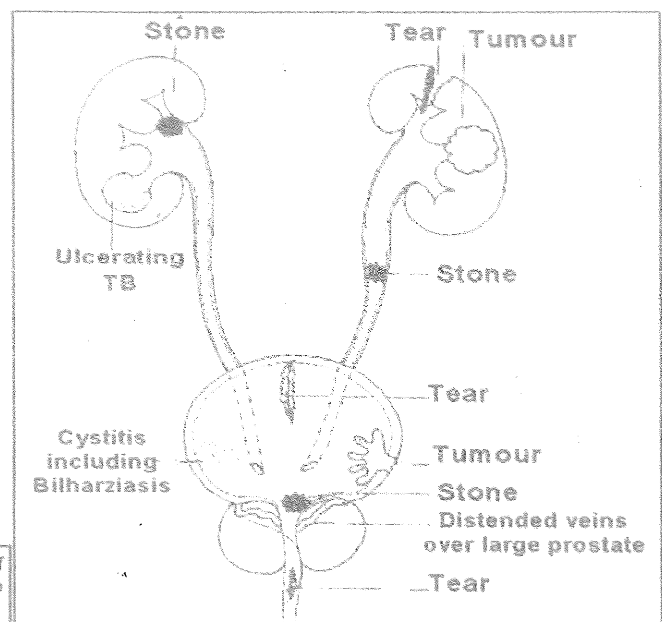


Fig. 42.10. Senile prostatic enlargement (SPE) is the most frequent cause of chronic retention. Residual urine collects in the post-prostatic pouch.



Fig. 42.11. Common causes of haematuria. Stone disease is the most frequent.



Haematuria

Definition

Haematuria means passage of blood in urine.

Types

- Frank or microscopic.
- Painful or painless. Painless haematuria without other symptoms "silent" must be regarded as a symptom of tumour until proved otherwise. It is usually intermittent.
- In relation to urine stream.
    - Total haematuria. Passage blood all over the stream. It indicates that blood comes from the kidney or from massive vesical bleeding. Haematuria from the kidney is usually associated with cylindrical clots. Haematuria from the bladder is associated with discoid clots. Stones, Senile prostatic enlargement and tumours are common causes.
    - Terminal haematuria. Passage of blood at end of rnicturition. It indicates a pathology in the trigone, bladder neck, posterior urethra and sometimes senile prostatic enlargement (prostatic piles). Bilharsiasis is a common cause.
    - Initial haematuria. Passage of blood at the beginning of the act. It indicates a urethral origin.

Aetiology

General causes

- Haemorrhagic blood diseases, e.g. thrombocytopenic purpura or haemophilia.
- Anticoagulant therapy.
- Renal causes
    - Renal stones.
    - Congenital causes, e.g., polycystic kidney.
    - Inflammatory diseases. Acute glomerulonephritis and tuberculosis.

- Traumatic. Renal trauma may be accidental, post-operative or pos instrumental.
- Neoplasms of the kidney. Hypernephroma, transitional cell carcinoma an Wilm's tumour.
- Vascular causes, e.g., renal infarction.

- **Ureteric causes**
  - Ureteric stones.
  - Surgical and instrumental procedures to the ureter.
  - Neoplasm of the ureter. Transitional cell carcinoma.
- **Bladder causes**
  - Inflammation. Non-specific cystitis, Bilharzial cystitis, or tuberculous cystitis.
  - Urinary bladder stones.
  - Neoplasms of the bladder.
- **Prostatic causes**
  - TB prostatitis.
  - Senile prostatic enlargement.
  - Prostate cancer.
- **Urethral causes**
  - Urethritis.
  - Urethral injuries.
  - Urethral stones.
  - Neoplasms of urethra.
- **The most common causes of haematuria** (Fig. 42.11)
  - Urinary Stones.
  - Urinary tract injuries.
  - Senile prostatic enlargement.
  - Renal tumours.
  - Bladder tumours.
  - Bilharziasis.

Differential diagnosis

Other causes of red colored urine are:

1. Dietary. The intake of red beetroots and the wide use of coloring agents in cakes, col drinks and fruit juices makes children commonly pass red urine after ingestion of these products.
2. Drugs, e.g., furadantin, rifampicin and laxatives that contain phenolphthalein.
3. Hemoglobinuria, which is the passage of haemoglobin in urine. It occurs with haemolytic states.

CONGENITAL ANOMALIES OF THE URINARY TRACT

Congenital anomalies of the urinary tract are fairly common. They range in severity from the very harmless that can be safely inored to the progressively damaging that ends in renal failure.

CHAPTER CONTENTS

Development of the urinary tract

Kidney, pelvicalyceal system and ureter

- A ureteric bud arises from the lower end of the mesonephric (Woliffian) duct and grows upwards retroperitoneally. This bud will form the ureter and its upper end will form the pelvis, the calyces and the collecting tubules (excretory elements).
- The upper end of the ureteric bud will be capped by the metanephros which will form the glomeruli and nephric tubules (secretory elements). Later, the nephric tubules establish continuity with the collecting tubules.
- Ascent and rotation. The kidney starts its development in the pelvis and later ascends to the loin. As the kidney ascends it also rotates. At first the pelvis lies anteriorly and the calyces posteriorly. Later, the pelvis rotates to lie medially while the calyces face laterally.

Bladder and urethra

Urinary bladder

The bladder develops from the urogenital sinus (anterior division of cloaca), allantois and Wolffian ducts. The latter form the trigone, ejaculatory ducts and vase differentia.

Urethra

- The whole urethra in females and posterior urethra in males are formed from the urogenital sinus.
- The anterior urethra in males develops from the inner genital folds.
- The glandular urethra develops by invagination of epithelium from the tip of glans penis.

Anomalies of the kidney

Classification

- **Anomalies of number**
    - Agenesis: Bilateral or unilateral.
    - Supernumerary kidney.
- **Anomalies of volume and structure**
    - Hypoplastic kidney.
    - Simple renal (solitary) cyst.
    - Polycystic kidney.
    - Multicystic kidney.
    - Medullary sponge kidney.
- **Anomalies of ascent**
    - Ectopic (pelvic or iliac) kidney.
- **Anomalies of shape, form and fusion**
    - Horse-shoe kidney.

- Discoid (cake-shaped) kidney.
- Sigmoid and L-shaped kidney.

- **Anomalies of rotation**
    - Incomplete rotation.
- **Anomalies of the renal pelvis**
    - Bifid pelvis (double pelvis).
    - Pelvi-ureteric junction obstruction.
- **Anomalies of renal vasculature**
    - Aberrant and accessory renal arteries (vessels).
    - Renal artery aneurysm and A-V fistula.

Simple renal (solitary) cyst

Aetiology

It is not clear whether this lesion is congenital or acquired. Its origin may be similar to that of polycystic kidneys, or it may be related to tubular obstruction.

Clinical features

1. A solitary cyst is commonly asymptomatic.
2. Intermittent dull pain in flank or back.
3. Hemorrhage in a cyst, or infection will lead to acute symptoms.
4. Renal mass if the cyst is huge (Fig. 43.1).

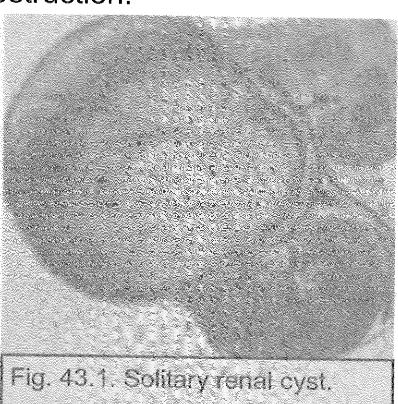


Fig. 43.1. Solitary renal cyst.

Differential diagnosis

Renal cysts should be differentiated from neoplastic renal masses. Although both lesions are space occupying on excretory urography, it is usually possible to distinguish between them by ultrasonography and CT scanning.

Simple renal cysts require no treatment.

Treatment

1. No treatment but follow up is recquired.
2. If the cyst causes hydronephrosis from renal or ureteric compression treatment is by aspiration of the cyst fluid and replacement with a sclerosing fluid such as 95% alcohol. Marsupialisation or excision may be recquired.
3. Atypical cyst (haemorragic cyst, thick wall or cloudy fluid). The following options are considered:
    a. Percutaneous needle spiration of content for analysis. Fluid is examined for colour, clarity, chemically and cytologically. Blood, high fat content or positive cytology, gives high suspicion of malignancy.
    b. Surgical exploration to excise the extrarenal portion of the cyst (Kirwin's method).
    c. Partial nephrectomy may be considered in rare cases.

Polycystic kidney

Adult type (autosomal dominant) polycystic kidney disease:

- This is the common form of cystic disease of the kidney.
- It affects both kidneys.
- It is one of the leading causes of end stage renal failure.

- Almost 50% have associated cysts in the liver, but liver function remains normal. Cysts of lung, pancreas and other organs may be found. 30 to 40% of patients have intracranial aneurysms.

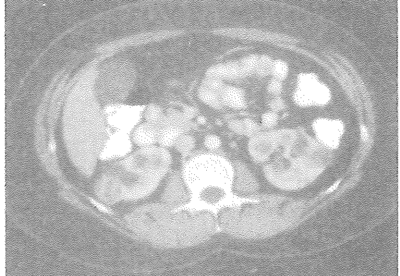

Fig. 43.2. Gross picture of a polycystic kidney.

Pathology

Cysts do not form before the age of 10 years. Grossly, the kidneys are usually enormously enlarged. Their surfaces are studded with cysts of various sizes (Fig. 43.2). On section, the cysts are scattered throughout the parenchyma. The cyst fluid is usually amber clear, but may be hemorrhagic or infected.

Clinical features

- For the first 10 years of life there are no symptoms because the kidneys are normal in function and anatomy.
- From 10 to 30 years, ultrasound show the presence of cysts, although the patient is still asymptomatic.
- After 30 years, the patient may develop microscopic or gross haematuria, hypertension, urinary tract infections and flank pain. The kidney may be palpable.
- Renal function impairment with elevation of urea and creatinine begins between age 40 to 50.
- Renal failure may occur after the age of 50.


Fig. 43.3. CT scan showing polycystic kidneys.

Investigations

- Urine examination. RBCs or pus cells may be present.
- Blood urea and serum creatinine are elevated.
- Ultrasonography or CT scan (Fig. 43.3) is accurate in the diagnosis of polycystic kidneys.
- Excretory urography with infusion shows enlarged renal shadows and bizarre calyceal pattern (spider leg appearance), as the calyces are stretched over the cysts (Fig. 43.4).


Fig. 43.4. IVU showing the spider-leg appearance of a polycystic kidney.

Treatment

Before renal failure

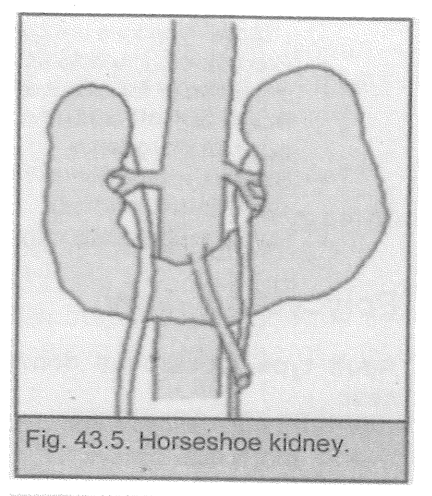
1. Control of hypertension by antihypertensive medications.
2. Treatment of UTI by appropriate antibiotics.
3. Low protein diet and regular check of renal functions.
4. If a large cyst compresses the pelvis or the upper ureter treatment is by percutaneous ultrasound-guided aspiration.

After renal failure

1. Renal transplantation is the definitive treatment.
2. Haemodialysis is a temporary measure until a suitable donor is found.

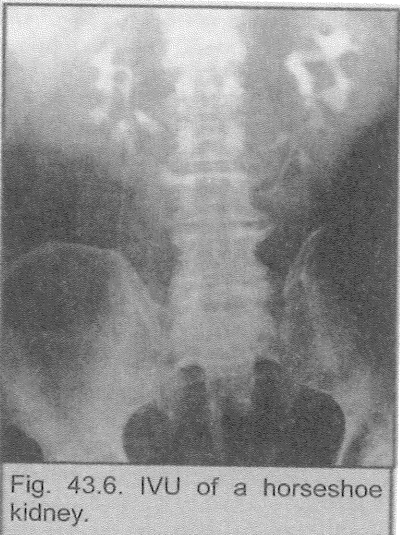
Horseshoe kidney

More frequent in males.


Fig. 43.5. Horseshoe kidney.

Pathology

- Fusion occurs early in embryologic life when the kidneys lie low in the pelvis.
- Ascent of the horseshoe kidney is arrested by the isthmus being blocked against the inferior mesenteric artery (Fig. 43.5).
- Furthermore, normal rotation cannot occur, and each renal pelvis lies on the anterior surface of its kidney. The ureters thus ride over the isthmus which connects the lower poles.
- A horseshoe kidney is prone to disease because ureteric obstruction may result from angulation of the ureter as it crosses renal isthmus. Stasis favours infection and stone formation.


Fig. 43.6. IVU of a horseshoe kidney.



Clinical features

- One third of patients remain asymptomstic.
- The rest develop symptoms of complications as pain, haematuria or fever.
- A hydronephrotic horseshoe kidney may be palpable below the umbilicus. A very serious mistake is to excise this undiagnosed mass in an exploratory laparotomy. Before excising such a mass always check for the presence of the kidneys by ultrasound or by IVU.

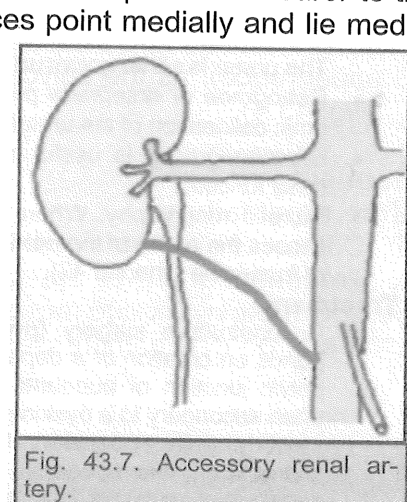
Investigations

IVU reveals that the kidneys are lower in position, the lower poles are nearer to the midline than the upper poles and that the lower pole calyces point medially and lie medial to the ureter (Fig. 43.6).

Treatment

Treatment is indicated only for complications. Division of the isthmus of the kidney is rarely required.

Aberrant and accessory renal vessels


Fig. 43.7. Accessory renal artery.

- A single renal artery is noted in 75-85% of individuals.
- Aberrant arteries originate from vessels other than the aorta, or main renal artery. They are very rare.
- Accessory arteries are those arising from the aorta, or main renal artery (Fig. 43.7).
- If one of these arteries passes to the lower pole of the kidney it may obstruct the ureter and produce hydronephrosis.
- Division of an accessory artery will cause infarction of the corresponding portion of renal parenchyma (end arteries). On the other hand, an aberrant renal vein can be divided without any ill effects.

Idiopathic pelvic-ureteric junction (PUJ) obstruction

- PUJO is a common cause of hydronephrosis in children and adolescents.
- It is more often seen in males with the left side predominating.
- Bilateral obstruction occurs in at least 10 to 15% of cases.

Pathology

- For an unknown reason there is failure of relaxation of the PUJ.
- Obstruction to urine outflow produces hydronephrosis which may either progress or reach an equilibrium.

Clinical features

- The clinical presentation varies according to age:
- Prenatal. Ultrasonography can diagnose the condition in the foetus.
- In infants the most frequent finding is an abdominal mass.
- In children episodic intermittent flank pain which follows fluid intake. Vomiting may be present.
- In adults loin pain, recurrent urinary tract infections and hypertension.

- **The kidney is the commonest urinary organ to have anomalies.**
- **Most renal anomalies are asymptomatic and need no treatment.**
- **The anomalous kidney is liable to any disease that affects a normal kidney.**
- **Polycystic kidney is a common cause of renal failure.**
- **PUJO and posterior urethral valves are common anomalies that require early treatment in order to preserve renal function.**

Investigations

- Excretory urography shows a dilated pelvicalyceal system with abrupt contrast arrest at the pelviureteric junction (Fig. 43.8). The ureter is either nonvisualized or of normal caliber.
- Retrograde or antegrade pyelography may be used for anatomic delineation of the ureter and pelvis.
- Ultrasonography is useful in advanced conditions with poor renal function.
- Diuretic renography. When the diagnosis is equivocal, It assesses the ability of the pelvis to empty after the administration of frusemide (chapter 42).

Fig. 43.8. IVU showing left idiopathic PUJO.

Traetment

1. Reconstructive surgery (pyeloplasty). Successful repair depends on creation of a dependent and funnel-shaped ureteropelvic junction of adequate calibre. Since the obstruction is often secondary to a dyskinetic segment of proximal ureter, the most popular operation is a pyeloplasty (Anderson-Hynes operation). This entails excising the proximal segment of ureter as well as the redundant part of the pelvis. The pelvis and PUJ are reconstructed in such a way to ensure good drainage (Fig. 43.9).
2. Endoscopic endopyelotomy may be considered in some patients specially those with previous failed surgical repair.
3. Nephrectomy is considered for a hopeless kidney with total renal function loss.

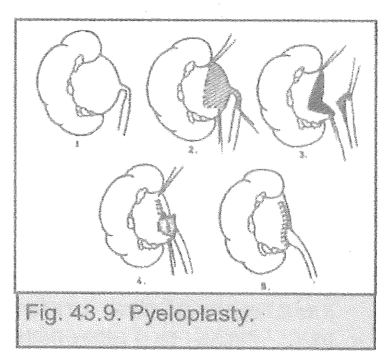


Fig. 43.9. Pyeloplasty.

Prognosis

Prognosis of PUJO correlates inversely with age, duration and severity of obstruction.

Anomalies of the ureter

Classification

1. Anomalies of number. Agenesis and duplication.
2. Anomalies of structure. Atresia and hypoplasia, megaureter, ureteral stenosis, ureteral valves and ureteral diverticula.
3. Anomalies of position. Retrocaval ureter.
4. Anomalies of termination. Vesicoureteric reflux, ectopic ureter, ureterocele,

Anomalies of the bladder

1. Bladder exstrophy (ectopia vesicae).
2. Persistent urachus. It leads to a urinary fistula at the umbilicus.
3. Contracture of the bladder neck.



Fig. (43.10): Ectopia vesicae

Bladder exstrophy (ectopia vesicae)

Incidence. One in 50,000 live births. It is much more common in males *(4:1)*.

Embryological basis. Exstrophy epispadias complex is due to failure of cloacal membrane to be reinforced by ingrowth of mesoderm. This leads to a ventral defect of urogenital sinus and overlying skeletal system.

Clinical features (Fig. 43.10)

- Lower part of anterior abdominal wall and anterior wall of the bladder are deficient.
- Inner surface of posterior wall of the bladder bulges through the defect with its mucosal edges fused with the surrounding skin.
- Urine spurts onto the abdominal wall from the exposed ureteral orifices.



Fig. (43.11): Pain X-ray of ectopia vesicae showing wide separation of the symphysis pubis

- Bladder mucosa appears normal at birth, but persistent irritation and recurrent infections lead to chronic inflammation, metaplastic changes, and sometimes malignant transformation.
- Pronounced widening of symphysis pubis (Fig. 43.11) which is connected by an intersymphyseal band. This leads to a characteristic waddling gait when the child begins to walk.
- An umbilical hernia is always present, and may be associated with oblique inguinal hernias as well.
- Distorted pelvic anatomy may contribute to varying degrees of anal incontinence and rectal prolapse.
- In males, there is associated epispadias with a short widened penis. The testes frequently appear undescended although most are actually retractile.
- In females, the clitoris is cleft, and there may be associated vaginal anomalies.

Complications

1. Recurrent urinary tract infections.
2. Excoriation of the skin of the anterior abdominal wall.
3. Sterility.
4. Development of bladder cancer due to metaplastic changes.

Treatment

- **Complete reconstruction** is now advisable.
    - The most important factor is early management. Exstrophy can now be diagnosed in utero by the characteristic ultrasound picture of a low set umbilicus, and repeated failure of the bladder to fill.
    - At birth the bladder mucosa should be covered with a nonadherent film to prevent unnecessary trauma. The primary objective in functional closure is to convert the exstrophy to a complete epispadias with incontinence while preserving renal function.
    - Within the first 48 hours, closure of the bladder and pelvis is easy, but if the patient is not seen until several days of age or older, then anterior or posterior pelvic osteotomy will be required to achieve closure.
    - Penile and urethral reconstruction is then attempted at 2 to 3 years of age, meanwhile correction of incontinence and reflux is even further delayed until bladder capacity has increased.
- **Urinary diversion** (uretero-sigmoidostomy) with excision of the bladder, followed later by repair of epispadiac penis was the usual line of management. However, because of the long-term problems associated with diversion, this policy is now reserved for delayed presentation or complicated cases.

Anomalies of the urethra

Hypospadias

Hypospadias means that the urethral meatus opens on the ventral aspect of the penis at any point from glans penis to the perineum. This anomaly is caused by incomplete development of the terminal part of the urethra and the corpus spongiosum. The missing distal part of the urethra is replaced by a fibrous band (chordee). Incidence One in every 300 male children have hypospadias. Positive family history is present in 8% of cases.

Aetiology

1. Hormonal causes. Deficiency of androgens or 5-alpha-dehydrotestosterone reductase enzyme during intrauterine life.
2. Deficient receptors in target cells may play a role.

Classification (Fig. 43.12)

1. Glanular. Meatus is at the glans.
2. Coronal. Meatus is at the coronal sulcus.
3. Penile. Meatus is at the ventral surface of the penile shaft (anterior, mid or posterior penile).
4. Penoscrotal. Meatus is at the junction of penis and scrotum.
5. Perineal. Scrotum is usually bifid and urethra opens between its two halves.

Hypospadias is anterior in 65% of cases, midpenile in 15%, and penoscrotal or perienal in 20% of cases.

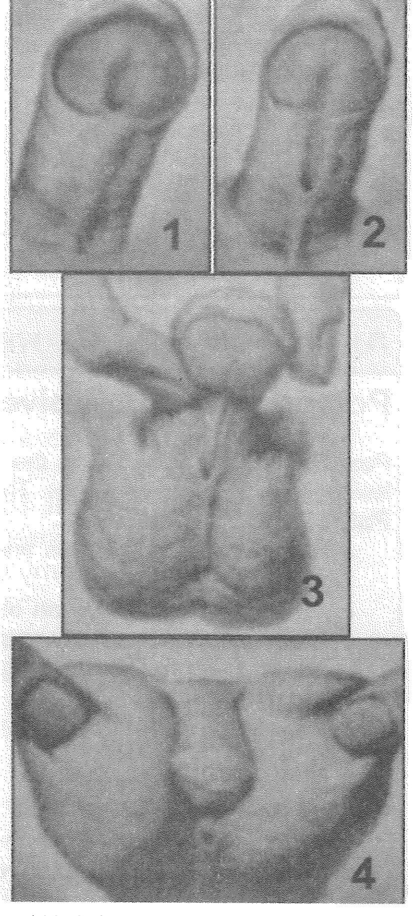


Fig. (43.12): Sme types of hypospadias
1. Glanular
2. Mid-shaft
3. Penoscrotal
4. Perineal

Clinical features:

- Meatus is ventrally placed and stenosed.
- Prepuce is deficient venterolaterally.
- Shaft is ventrally curved due to the presence of chordee except in the glanular variety.
- Scrotum is bifid in the perineal type.
- Associated lesions
    - 10% of patients have inguinal hernia or undescended testes.
    - 8% of patients have upper urinary tract anomalies.
    - Patients with posterior hypospadias may have a problem wfth sex differentiation.

Investigations

Ultrasound examination to detect upper urinary tract problems.

Treatment

- Circumcision should not be done to hypospadiac patients becuase the skin of the prepuce can be used for repair.
- Plastic surgery can be performed at the age of one year. The aim of surgery is to have a normally functioning male organ with normally situated meatus at tip of the glans.
- The principle of surgery is to release the chordee so that the ventral curvature of the penis is corrected. Then a new urethra is fashioned using neighbouring skin from the prepuce or the penile skin.
- One stage surgery can be done in most cases. However, in difficult or severe cases, two-stage surgery may be done.

Epispadias

Definition

Epispadias is the reverse of hypospadias. The urethral meatus opens on the dorsum of the penis.

Incidence

Complete epispadias occurs is approximately 1 in 120,000 males and 1 in 450,000 females.

In males

Epispadias is classified according to the position of the dorsally displaced urethral meatus into glanular, penile (Fig. 43.13), penopubic and complete types. All are associated with varying degrees of dorsal chordee. Glanular epispadias is not associated with incontinence, but penile and penopubic epispadias are associated with incontinence

in 75 and 95% of cases respectively. Epispadias is a mild form of bladder exstrophy, and in severe cases both conditions coexist.

In females

Females with epispadias have a bifid clitoris, separation of labia, and most of them are incontinent

Treatment

Surgery is required to straighten the penis, and extend urethra to glans penis as well as to achieve continence. Continence further improves with growth of patient because of the effect of urethral lengthening and prostatic enlargement.

Fig. (43.13): Epispadias

INJURIES OF THE URINARY TRACT

A large number of injuries that are seen in the causality department involve the urinary tract. Urethral injuries are the most frequent of them.

The principles of management of trauma victims are outlined in chapter 2.

CHAPTER CONTENTS

Renal injuries

Incidence

- Injuries of the kidney are relatively rare due to:
  - The kidneys are well protected by the rib cage and by the heavy muscles of the back.
  - The kidneys are mobile in their adipose fat and thus flee away from the force of trauma.
  - The fibrous capsule does protect the parenchyma from splitting.
- Renal injuries are potentially serious and may be complicated by injuries of other organs.

Aetiology

1. Blunt injuries are the commonest (80-85%) due to:
   a. Direct trauma to the abdomen (common), flank or back e.g. car accidents.
   b. Indirect (less common) or contre coup injury, e.g., falling from a height and landing on the feet or buttocks.
2. Penetrating wounds e.g. knives or bullets are rare. Associated visceral injuries are present in 80% of renal penetrating wounds.
3. Iatrogenic injuries occur during surgery.

Pathology

In contrast to intraperitoneal organs, bleeding from renal injuries is confined to the retroperitoneal space, which usually stops further bleeding by virtue of its tamponade effect.

Types of injury (Fig. 44.1)

1. Bruising and ecchymosis are the most frequent lesions.
2. Subcapsular haematoma.
3. Laceration. These usually occur in the transverse axis of the kidney or radiate from the hilum.
   a. If a laceration extends through the capsule a perinephric haematoma results.
   b. If it reaches the pelvicalyceal system, haematuria occurs, with or without perirenal urine collection.
4. Vascular injuries
   a. Rupture of the whole renal vascular pedicle causes massive bleeding (about 1% of blunt trauma cases), and is an emergency.
   b. Avulsion of the renal artery or vein or avulsion of a segmental branch.
   c. Stretch of the main renal artery leads to an intimal tear with thrombosis.
5. Associated injuries:

a. Tear in the peritoneum with intraperitoneal bleeding.
b. Visceral injuries.
c. Fracture of the spine or ribs.

Complications

1. Hydronephrosis. Fibrosis around kidney due to extravasation of urine and blood leads to ureteric obstruction and hydronephrosis.
2. Renal atrophy or fibrosis. Impaired blood supply by rupture, thrombosis or fibrosis leads to atrophy.
3. Renovascular hypertension may develop from renal ischaemia.
4. Perirenal cyst. If haematoma becomes infected or liquified, a localized collection of clear amber fluid may remain.
5. Arteriovenous fistula may occur after penetrating injuries.
6. Pseudohydronephrosis. Accumulation of urine all around the kidney.

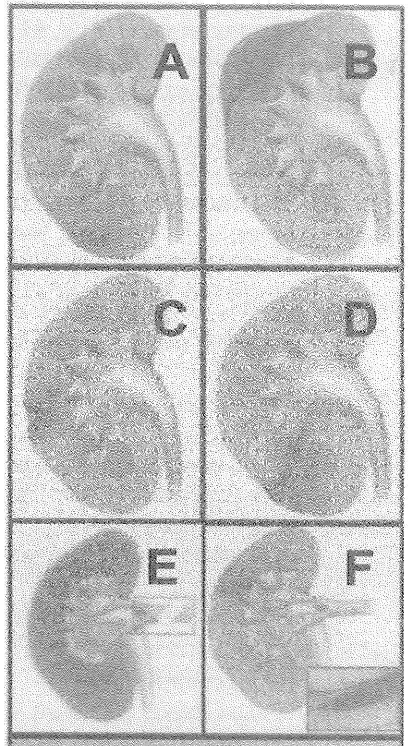


Fig. 44.1. Types of renal injuries.
A. Contusion..
B. Subcapsular haematoma.
C. Laceration not reaching pelvicalyceal system.
D. Laceration reaching pelvicalyceal system.
E. Avulsion of renal artery.
F. Injury of a segmental artery.

Clinical features

Symptoms

1. History of trauma.
2. Pain localized to the renal area. This pain may be obscured by injury to other organs, e.g., fractures or injury to viscera causing diffuse abdominal pain.
3. Haematuria following trauma indicates urinary tract injury. Its degree is not necessarily proportionate to the severity of injury. Haematuria is usually noted with the first voiding, but may be late to appear due to hypotension. It may be continuous or intermittent. About 30% of vascular injuries are not associated with haematuria. Haematuria will be absent if there is complete avulsion of the pedicle or if there is a ureteric injury.
4. Nausea, vomiting and abdominal distension due to intestinal atony are common in the presence of retroperitoneal bleeding.
5. Oliguria due to hypovolaemia and hypotension.
6. Retention of urine due to clots in the bladder.

Signs

1. Haematuria of variable degree in collected urine samples.
2. Signs of haemorrhage and probably shock in severe renal trauma or multiple injuries.
3. Local tenderness, guarding and rigidity on the affected side with rebound tenderness may be found.
4. A mass in the flank may be present due to extravasated blood and urine. If the overlying peritoneum has been torn, it will leak into the peritoneal cavity causing diffuse abdominal tenderness, abdominal distension and hypoperistalsis.

Investigations

Laboratory

1. Urine analysis. Red blood cells are present in almost all cases.

2. Haematocrit value. Progressive anaemia means progressive haemorrhage.
3. Renal function tests may be impaired from prolonged hypotension, bilateral injuries or injury of a solitary kidney.

Imaging

Ultrasound confirms the presence of renal injury. It can show the size, and contour of the kidney and can visualize a collection around it.

Plain UT (X-ray film) does not show the injury but may show indirect evidence, e.g., obliteration of psoas shadow by a haematoma. It also shows associated rib or spine fractures.

I.V.U. Infusion urography is done to visualize the upper urinary tract as soon as the shock is controlled and the blood pressure is above 100mm Hg. It may show:

- Normal function and configuration of kidney if the injury is minimal.
- Deformed renal pelvis or calyces if there is laceration or blood clots.
- Extravasation of contrast within the renal shadow or into perirenal space (Fig. 44.2).
- Non-visualization of kidney due to total pedicle avulsion, arterial thrombosis or severe contusion causing vascular spasm.
- Confirms the presence of a functioning kidney on the opposite side, as nephrectomy of the injured kidney may be needed.

Fig. 44.2. Injured right kidney showing extravasation of contrast into perirenal space on an IVU.

CT scan shows parenchymal lacerations, urinary extravasation, perirenal haematoma, non-viable renal tissue, as well as other organ injuries. It is more accurate than ultrasound in assessing the injury.

Angiography is needed in cases suspected to have renal pedicle injury.

Renal isotope scanning (DMSA) is useful in patients who are allergic to contrast material. Lack of uptake implies injury or laceration of the renal artery. Areas of decreased activity are compatible with renal contusion, while absence of uptake in one pole suggests amputation. Perirenal extravasation of radioactive urine may appear.

Treatment

Renal trauma is an acute emergency. Most renal injuries will be cured by conservative management, as most injuries (85%) are minor. The principles of trauma victims care (chapter 2) should be followed.

Conservative treatment

- Hospitalization with bed rest until haematuria has ceased and local signs of injury have subsided.
- Analgesics for pain.
- Large fluid intake to guard against clot retention and for hypovolaemia.
- Broad spectrum antibiotics to guard against secondary infection.
- Follow up parameters
  - Pulse, blood pressure, and size of any perirenal mass.
  - Repeated samples of urine are examined and compared grossly for red colour. Repeated urine analysis for RBCs.
  - Haemoglobin and haematocrit estimations.

Surgery

Indication

1. Persistent progressive haematuria or failure to stabilize vital signs.
2. Presence of a progressively enlarging perirenal mass.
3. Evidence of perirenal infection.
4. Penetrating injuries.
5. Renal pedicle injury (5% of all renal injuries).
6. Presence of an associated intraperitoneal injury.
7. Indications for delayed surgery
    a. If hydronephrosis develops, it is treated by relief of obstruction.
    b. If hypertension develops, vascular repair or hephrectomy is performed.

Principles

1. Transperitoneal approach.
2. The colon is reflected medially.
3. Preliminary control of the vascular pedicle using vascular clamps.
4. A haematoma around the injured kidney is evacuated.
5. Traumatized and devascularized renal tissue is debrided.
6. Small defects of cortical tissue are approximated by sutures. Large defects are filled by omental or perirenal fat to obliterate dead space.
7. Water-tight closure of the pelvicalyceal system.
8. Partial nephrectomy is done if one pole of the kidney is avulsed.
9. Nephrectomy is done if the kidney is shattered or with complete avulsion of vascular pedicle, provided that .the other kidney is functioning well.

Ureteric injuries

Aetiology

Iatrogenic injuries are the most frequent.

1. Blunt or penetrating trauma is relatively rare.
2. Surgical injury during difficult or extensive gynaecoloic operations or abdominoperineal resection of the rectum, in difficult operations preliminary ureteric catheterization helps exposure of the ureter early in the procedure.
3. Endoscopic urological procedures, e.g., cystoscopic ureteric catheterization, uretroscopy and nephroscopy.
4. After radiation therapy to the pelvis.

Pathology

The following consequences may take place.

1. Extravasation of urine which may be:
    a. Intraperitoneai, leading to peritonitis or to urinary fistula if a drain has been placed.
    b. Extraperitoneal, which appears as an enlarging collection of urine at the site of injury. A retroperitoneal collection can cause reflex paralytic ileus.
2. Ueteric obstruction, eg., by a ligature. Complete obstruction causes suppression of urine production from the corresponding kidney. Partial obstruction causes hydronephrosis,
3. Anuria and acute renal failure may complicate bilateral injuries or those affecting the only functioning kidney.

Clinical features

1. History of trauma or surgery.
2. Nausea, vomiting, loin pain and probably fever.
3. Oliguria or anuria in bilateral cases. Symptoms and signs of acute renal failure develops in neglected cases.
4. Urinary leakage or fistula.
5. Renal tenderness and/or renal mass (hydronephrosis).

Investigations

1. Ultrasound will reveal distension of the pelvicalyceal system in patients with ureteric obstruction.
2. Intravenous pyelography shows distension of pelvicalyceal system, delayed secretion or extravasation of the contrast.
3. Ureteric catheterization and retrograde pyelography will reveal obstruction and extravasation at the site of injury.

Differential diagnosis

1. Post-operative distension and peritonitis.
2. Post-operative acute pyelonephririts
3. Post-operative drainage of peritoneal fluid.

Complications

1. Stricture formation with hydronephrosis.
2. Ureteric urinary fistula.

Treatment

Management of ureteric injuries is essentially surgical and depends upon the general condition of the patient, extent and site of injury and time of presentation. The principles of repair are debridment, tension-free spatulated anastomosis, water-tight closure, uretral stenting and drainage.

1. Exploration of the ureter and repair of injury by end-to-end anastomosis or by reimplantation of the lower end of ureter into the bladder. This is indicated if the general condition of the patient is good, surgeon is experienced and injury is discovered early (within the first week).
2. Urinary diversion by a percutaneous nephrostomy and postponing ureteric reconstruction is the safest procedure in unfavourable circumstances.
3. Ureteric catheterization and stenting may be successful in some cases.

Urinary bladder injuries

Bladder Injuries occur most often from external force, and are frequently associated with pelvic fractures. Bladder injury is either extraperitoneal (80%) or intraperitoneal (20%) according to which part of bladder is injured. Sometimes, both injuries are present.

Aetiology

1. Fracture of the pelvis is the commonest cause of extraperitoneal rupture where a spike of bone injures the extraperitoneal surface of the bladder.
2. A blow or a kick to the lower abdomen, in the presence of a full bladder, is the commonest cause of intraperitoneal rupture where the bladder dome ruptures posteriorly.

3. Open injuries caused by stabs or bullets.
4. Surgical operations as hernia repair where there may be a sliding bladder, hysterectomy or abdomino-perineal resection.
5. During cystoscopic procedures as transurethral resection of the prostate or diathermy fulguration of a bladder tumour.

Clinical features

Extraperitoneal rupture (Fig. 44.3)

1. History of serious injury which caused pelvic fracture.
2. Hypovolaemic shock.
3. Urine starts to collect in the retropubic space giving rise to an intense desire to void, but the patient may pass only a few drops of blood-stained urine or none at all.
4. A boggy swelling appears in the suprapubic area and rises upwards as urine extravasates between the peritoneum and the transversalis fascia (similar to intrapelvic rupture of the urethra).
5. Digital rectal examination reveals the prostate in its normal position (to differentiate it from intrapelvic rupture of the urethra).
6. If the condition is not treated, chemical irritation of the anterior abdominal wall will occur. A necrotizing phlegmon will develop.

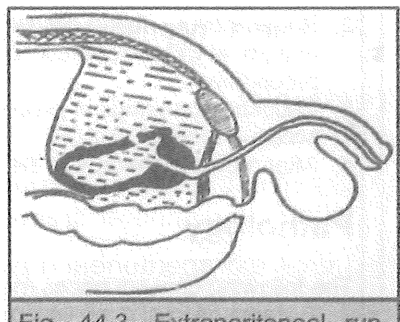


Fig. 44.3. Extraperitoneal rupture of the urinary bladder.

Intraperitoneal rupture (Fig, 44.4)

1. Sudden agonizing pain in the suprapubic area, which may be accompanied by shock. This pain is later replaced by a dull aching pain spreading all over the abdomen.
2. Severe oliguria or anuria as urine collects in the peritoneal cavity.
3. Peritonism and later peritonitis with tenderness, rebound tenderness and guarding spreading all over the abdomen. Abdominal distension is present, and if the amount of urine in the peritoneal cavity is large, shifting dullness can be elicited.
4. Digital rectal examination may reveals fullness in rectovesical pouch.
5. Passing a urinary catheter brings no urine.

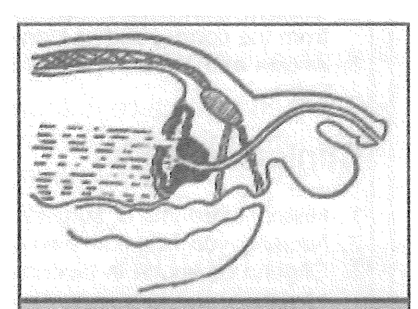


Fig. 44.4. Intraperitoneal rupture of the urinary bladder.

Investigations

1. Plain X-ray may show pelvic fracture and hazziness over the lower abdomen.
2. I.V.U excludes other urinary injuries and lack of filling of urinary bladder.
3. Ascending cystogram provides a definite diagnosis by demonstrating leakage of contrast outside the bladder (Fig. 44.5).



Fig. 44.5. Ascending cystogram showing intraperitoneal leakage of contrast.

Differential diagnosis

Intrapelvic complete rupture of the urethra should be differentiated from extraperitoneal rupture of the bladder. In the former, the prostate migrates up from the pelvis and is felt higher than normal on digital rectal examination.

Complications

1. Pelvic abcess from infection of a haematoma or urine collection.
2. Delayed peritonitis.
3. Partial incontinence if bladder neck is injured.

Treatment

- Management of shock.
- Small extraperitoneal rupture with minimal extravasation on cystogram may be treated with Foley catheter drainage for two weeks till healing occurs.
- Large extraperitoneal rupture and intra-peritoneal rupture need repair.
- The bladder tear is exposed, its edges are trimmed and the defect is closed in two layers with polygalactin (vicryl) or chromic catgut. A Foley catheter is left in the bladder and a drain is placed in retropubic space. In cases of intraperitoneal rupture, the peritoneum has to be opened to drain extravasated urine and to exclude any associated intraperitoneal injury.
- Antibiotic prophylaxis.
- The pelvic fracture is then treated according to its type. It should be noted that internal fixation of the broken bone is contraindicated in the presence of urine extravasation, for fear of causing osteomyelitis.

Urethral injuries

Urethral injuries are more common in men and are rare in women.

Sites

There are two common sites for urethral injuries (anterior and posterior, Fig. 44.6)

1. Anterior urethra (bulbous and penile). Bulbous urethral injury is more frequent than that of the pendulous penile part.
2. Posterior urethra (prostatic and membranous). Membranous urethral injury is more frequent than that of the prostatic part.

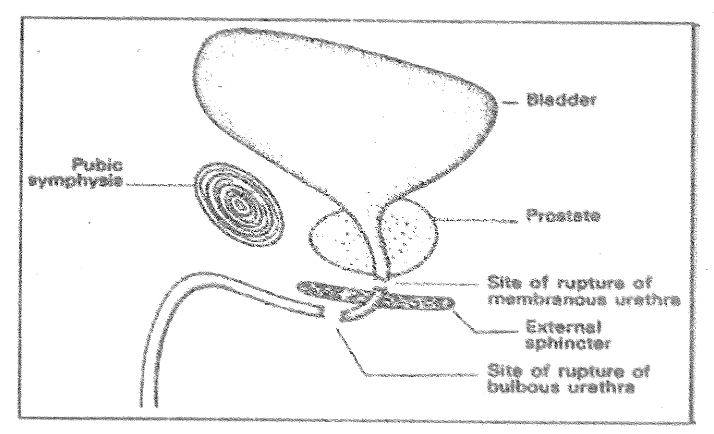


Fig. (44.6): Common site of urethral injury

Types

- Injury may be complete or incomplete, according to its extent on the circumference.
- Injury may be total or partial according to its depth in the wall.

Injury of posterior urethra (membranous urethra = intrapelvic rupture)

Aetiology

1. Severe trauma that causes pelvic fracture, e.g., road traffic accidents (similar to extraperitoneal rupture of the bladder). The urinary bladder and membranous urethra are sometimes simultaneously injured.
2. Iatrogenic injuries are usually caused by inexpert transurethral instrumentation.

Pathology

Types

1. Mucosal tear and false passage are caused by faulty instrumentation.
2. Laceration of a part of the circumference.
3. Complete circumferential laceration.
4. Complete circumferential laceration with torn pubo-prostatic ligament. The urinary bladder and prostate are thus free of any fixation and float upwards leaving a gap between the torn ends of urethra (Fig. 44.7).

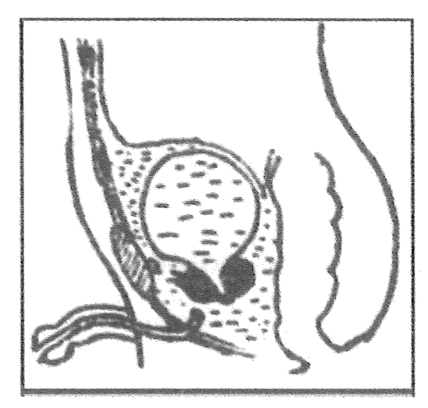


Fig. (44.7): Severe form of membranous urethra injury where the prostate is floating upwards. If the patient tries to urinate urine extravasates in a tissue plane similar to that of extraperitoneal rupture of the bladder

Complications

1. Blood loss and haemorrhagic shock are common.
2. Deep extravasation of urine in the extraperitoneal space. The condition is similar to extraperitoneal injury of the bladder.
3. Injury of external sphincter (sphincter urethrae). If the internal sphincter (sphincter vesicae) is also damaged, the patient will be incontinent.
4. Impotence may result because of injury of nerves to corpora cavernosa that course adjacent to the membranous urethra.
5. Urethral stricture. Long difficult strictures follow injuries that produce wide separation of torn ends.

Clinical features

- History of severe trauma. The patient may have hypovolaemic shock.
- The patient is usually unable to void urine though he feels the desire to micturate.
- Injury of membranous urethra should be suspected with pelvic fracture and when there are drops of blood at the external meatus.
- Digital rectal examination may reveal a higher than normal floating prostate.
- Distended urinary bladder excludes extraperitoneal rupture of urinary bladder.
- Whenever urethral injury is suspected catheterization of the bladder is contra-indicated as it may compound the damage and introduce infection.

Investigations

- Ascending urethrogram shows site of extravasation at prostatomembranous junction. The pelvic bones are assessed for the extent of fracture.
- Urgent intravenous urography (IVU) to detect associated urinary tract injuries and to see the position of the urinary bladder.

Differential diagnosis: Extraperitoneal bladder rupture.

Treatment

- Blood transfusion and other resuscitative measures are commonly required.
- A suprapubic cystostomy (Fig. 44.6) is performed in the emergency room, with no attempt at catheterization.
- The pelvic fracture is immobilized and the patient is later investigated for the development of urethral stricture.
- Treatment of urethral stricture later by either endoscopic visual urethrotomy or surgery.

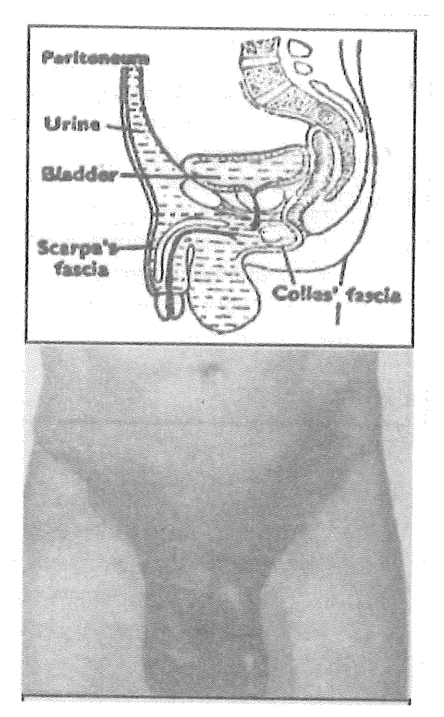


Fig. (44.8): Rupture of bulbous urethra. If the patient tries to micture superifical extravasation of urine occurs.

Injury of anterior (bulbous) urethra (extrapelvic rupture)

Aetiology

This injury is caused by a crushing force in the perineum.

- Falling astride a hard object, e.g., bar of a bicycle is a common cause, thus crushing the urethra against the pubis.
- A kick to perineum produces a similar effect.

Pathology

Type of injury

1. Contusion.
2. Laceration that does not involve the whole circumference.
3. Laceration that involves the whole urethral circumference.

Complications

1. Urine extravasation. If the patient tries to void, urine extravasates through superficial perineal pouch.
2. Stricture of urethra.
3. Urethral fistula.
4. Neglected cases get infected, and sloughing of perineal skin may occur.
5. Periurethral abscess.

Clinical features

1. History of classic trauma, perineal pain, and passage of few drops of blood from anterior urethral meatus.
2. Perineal haematoma.

3. Urine extravasation, if present, involves the perineum, penis, scrotum, and lower part of anterior abdominal wall (Fig. 44.8).
4. Urine can not be obtained.

The triad of urethral haemorrhage, a perineal haematoma, and retention of urine is diagnostic.

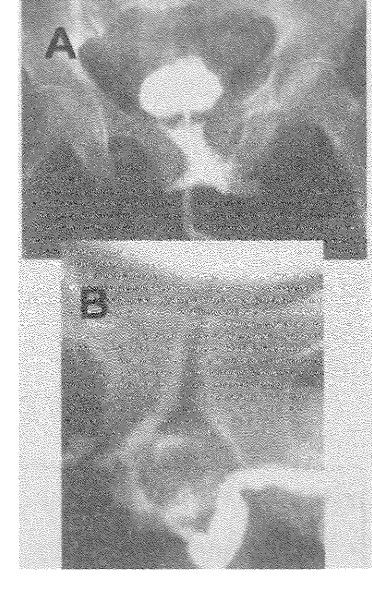


Fig. (44.9): Ascending cystograms showing superficial extravasation of contrast in the perineum. (A) In a partial circumference injury the bladder is filled. (B) In a complete injury the bladder does not fill.

Investigations

Ascending urethrogram shows the extravasation (Fig. 44.9).

Treatment

- If urethral injury is suspected, the patient is instructed not to try voiding.
- Passage of catheter is contraindicated as it can transfer a partial tear into a complete one and introduces infection.
- If there is no clinical extravasation, diagnosis of type of injury is obtained by an urgent ascending urethrogram.
- Immediate repair of the urethra is arely required.
- **Early treatment:**
  - Contusion of bulbous urethra usually needs no treatment.
  - If urethrogram shows extravasation of contrast material, diversion of urine by suprapubic cystostomy is urgently performed, and the patient is given prophylactic antibiotics.
  - An expanding haematoma of perineum or infected urine extravasation requires incisions and drainage.
- **Later treatment**. After three weeks the urinary bladder is filled with a contrast material and a micturating cystourethrogram is obtained.
  - A normal urethra is an indication for removal of the suprapubic tube.
  - If stricture develops, endoscopic urethrotomy followed by repeated dilatation are performed.
  - For tight and long strictures, surgical correction may be required.

Urological injuries
Points to remember
1. **Treatment of renal injuries is mainly conservative.**
2. **Most ureteric injuries are iatrogenic following difficult surgical and endoscopic procedures.**
3. **Bilateral ureteric ligation will lead to anuria.**
4. **The best way to avoid intraoperative ureteric injuries is to do preliminary exposure of the ureters.**
5. **Bladder or urethral injuries should be suspected in patients with pelvic fractures.**
6. **Extrapelvic injury of the urethra is diagnosed by the triad of; retention of urine, few drops of blood at the external meatus and perineal swelling.**
7. **Catheterization is contraindicated in patients suspected of having rupture of the urethra. The proper treatment is to do suprapubic cystostomy.**

URINARY TRACT INFECTIONS

The urinary tract may be divided into the upper tract, comprising ureter and kidney, and the lower tract, comprising bladder, prostate and urethra. Urinary tract infections are designated UTIs and can be discussed under two headings; non-specific and specific infections.

CHAPTER CONTENTS

Non-specific infections

General principles

Incidence

These infections are more frequent in females than males.

Aetiology & Pathology

Causative organisms

1. Escherichia coil (E. coli) in 80% of UTIs.
2. Proteus mirabilis (urea-splitting organism, produces ammonia and alkalinizes urine).
3. Pseudomonas aeruginosa (with urinary stasis).
4. Gram Positive Cocci (Staphylococci & Enterococci) are rare.
5. Chlamydia (with diabetes, prolonged antibiotic abuse and decreased immunity).

Route of infection

1. Ascending Infection is the most common route. Organisms that reside in the perineum; ascend through the urethra, especially in females.
2. Haematogènous spread is rare, It occurs with a perinephric abscess.
3. Lymphatic spread, from colonic and rectal lymphatics, is also rare.

Predisposing factors

1. Bacterial virulence.
2. Females, particularly the sexually active, are more prone to infection because:
   a. They have a short urethra.
   b. During sexual intercourse, bacteria are transferred from the vulvo-vaginal and urethral areas into the bladder.
   c. Pregnancy is associated with functional ureteric slow peristalsis, and hence a higher incidence of pyelonephritis.
3. Defective immunity, e.g., diabetes mellitus and immunosupression.
4. Urinary stasis, which may be mechanical as stones, or functional as neurogenic bladder.
5. Congenital anomalies, e.g., posterior urethral valves or vesico-ureteric reflux (VUR).
6. Urethral instrumentation may push bacteria from normally colonized anterior urethra

Types of UTI

- Uncomplicated UTI. There is no structural urinary tract abnormality and infection is usually harmless to the kidneys. A short course of an antibiotic eradicates the infection.

- Complicated UTI is accompanied by obstruction or stones. A combination of upper UTI and obstruction may lead to rapid renal damage. Complicated infection is often slow to respond to antibiotics, and often requires relief of obstruction.

Complications of UTI

1. Bacterial persistence and chronicity if not treated properly.
2. Septicaemia and septic shock.
3. Stone formation.
4. Impairment of renal function that may end in renal damage.

Clinical features

These features depend on:

- Whether it is upper or lower urinary tract infection.
- Whether infection is acute or chronic.
- The affected organ. Acute infections of the parynchymatous organ of the genito-urinary tract (kidney, prostate and testis) are usually associated with fever and need urgent treatment.
- Figure 45.1 shows a summary of the clinical features.

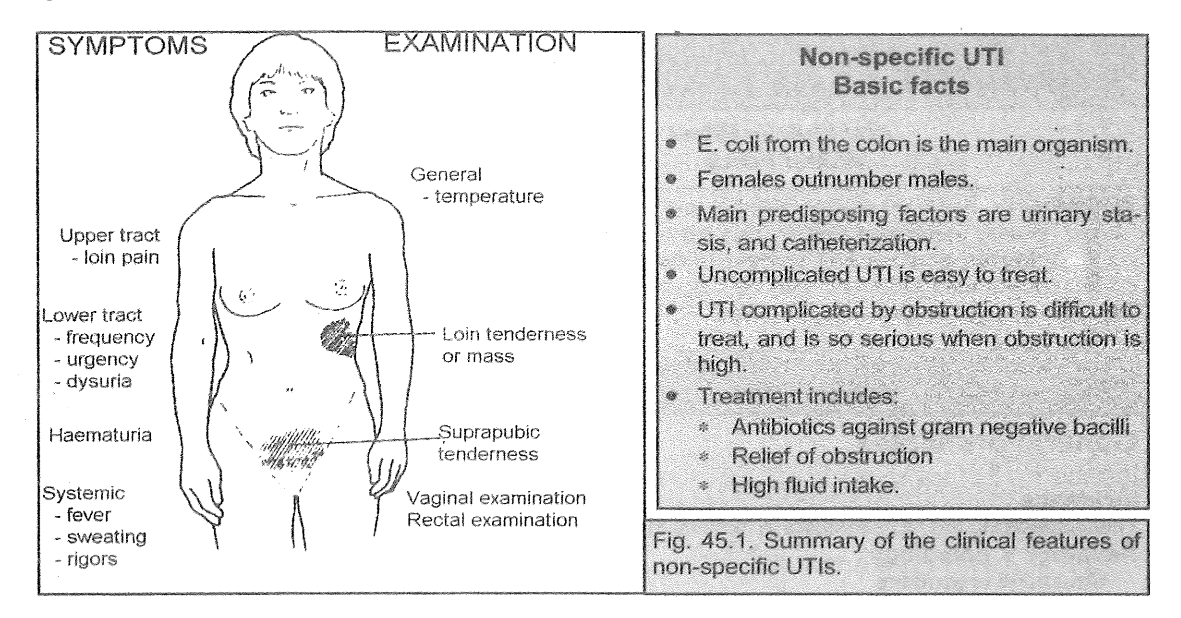


Fig. 45.1. Summary of the clinical features of non-specific UTIs.

Investigations

The aim of investigations is to:

1. Establish the diagnosis of infection; identify the organism and its antibiotic sensitivity.
   a. A clean catch mid stream urine (MSU) specimen is obtained and cultured immediately. In babies, a specially designed bag attached to genitalia is used.
   b. The finding of pus cells in urine (pyuria) is highly suggestive of infection.
   c. The microscopic finding of bacterial counts more than 10^5/ml is diagnostic.
2. Exclude structural and pathological abnormalities, e.g., stones. Investigations include plain X-Ray (KUB) and Ultrasound. IVU and urinary flow rate are needed in selected cases e.g.:
   a. Upper tract infections (e.g. pyelonephritis), specially if associated with fever.

b. Infections in children (to look for congenital anomalies).
c. Persistent bacteruria.
d. Recurrent infections.

Treatment

1. High fluid intake in order to induce diuresis.
2. Antibiotics are mandatory for all cases.
   a. **Route**. Oral antibiotic administration is used ior most cases. IV administration is required for severe cases of pyelonephritis with fever and rigors.
   b. Choice depends on culture and sensitivity; the most commonly used are trimethoprim/sulphamethoxazole (septrin), nitrofurantoin and quinolones.
3. Relief of obstruction, which may be urgent.
4. Drainage of an abscess, if present.

Prophylaxis for recurrent cases.

1. Fluid intake of at least 2 litres per day.
2. Perineal hygiene especially in females.
3. Regular voiding.
4. Avoid constipation, which may impair bladder emptying.
5. Prophylactic small doses of antibiotics may be used at bedtime for months.

Pyelonephritis (acute & chronic)

Pyelonephritis is infection of the renal parynchyma and pelvis. It may be acute or chronic. It is commoner in females particularly during pregnancy. A distinction, between a non-obstructed and an obstructed acute pyelonephritis is important.

Aetiology

E. coli is the usual organism but Proteus may be found. Infection is usually ascending, and less commonly haematogenous.

Complications

1. Septicemia and septic shock.
2. Pyonephrosis. Obstruction of urine flow in the acutely infected kidney produces dilatation and converts it into a bag of pus.
3. Renal damage. A combination of obstruction and infection rapidly damages the kidney.
4. Perinephric abscess.
5. Chronic pyelonephritis. Scarring of the kidney leads to:
   a. Renal hypertension.
   b. Chronic renal failure if the disease affects both kidneys.

Clinical features

Symptoms. Sudden severe loin pain, with fever, rigors and vomiting. Frequency, burning micturition and dysuria may be present.

Signs. Temperature rises up to 39-40°C. There is tenderness in the renal angle, and may be muscular rigidity. Bilateral pyelonephritis may give features of uraemia. In severe cases, the patient looks very ill and if not treated, it may lead to septicaemic shock.

Investigations

1. Mid stream urine catch (MSU) for routine analysis with culture and sensitivity testing.
2. Blood urea and serum creatinine.
3. Blood sugar and electrolytes.
4. Plain X-ray & IVU may show calcifications and kidney dilatation.
5. Ultrasound to pick up obstructed cases with dilatation of the pelvis and calyces.

Differential diagnosis of acute pyelonephritis

1. Basal pneumonia and pleurisy.
2. Acute cholecystitis. Ultrasound can differentiate.
3. Acute appendictis. The initial pain in appendicitis is around the umbilicus.
4. Acute pancreatitis.
5. Perinephric abscess.

Treatment

Non-obstructed acute pyelonephritis

A- General measures
   a. Rest in bed.
   b. Adequate hydration by drinking a large amount of fluids. If there is vomiting IV fluids and antiemetics are given.
   c. Analgesics and anti-pyretics as non-steroidal anti-inflammatory drugs.
   d. Alkalinization helps to relief dysuria and to inhibit growth of organisms.

B- Specific measures
   a. Antibiotics effective against Gram-negative bacilli are started until the culture and sensitivity result is available. Treatment should continue for 3 weeks.
   b. Urological investigations are done to exclude underlying abnormality. Repeated urine cultures should be done to ensure cure.

Obstructed acute pyelonephritis

- The same measures as the non-obstructed cases.
- Urgent drainage of the obstructed kidney by ultrasound-guided percutaneous nephrostomy until the patient improves and then the cause of obstruction is dealt with, e.g., percutaneous nephrolithotomy (PCNL) for a renal stone.
- Cystoscopy and ureteric catheterisation to drain the kidney may be done.

Pyelonephritis in children

Vesico-ureteric reflux (VUR) is the usual cause of pyelonephritis in children and is due to:

1. Idiopathic (primary) VUR: due to delay of maturation of uretero-vesical junction.
2. Secondary VUR that occurs on top of an infra-vesical obstruction such as posterior urethral valves, neuropathic bladder associated with spina bifida, or stones.

Fig. 45.2. Micturition cystourethrography shows left VUR. The contrast rushes up the left ureter during voiding.

Normally during voiding no urine passes upwards to the ureters or kidneys because bladder muscle contraction occludes the lower ends of both ureters Failure of this mechanism produces

VUR (Fig. 45.2). In most cases reflux is limited to the lower ureters and is self-correctable as the child grows up.

In a few cases, however, refluxing urine reaches the kidneys and the combination of high pressure generated by bladder contraction and infected urine produces renal damage. Repeated attacks of acute pyelonephritis cause scarring of the kidneys (chronic pyelonephritis), and probably renal failure. Uretero-vesical re-implantation with an anti-reflux technique may be required in some cases.

Pyonephrosis

Aetiology

Pyonephrosis is retention of infected urine and pus in the kidney. It may be:

1. Primary, due to coincident infection and obstruction, as in cases of renal calculi.
2. Secondary, due to infection of a pre-existing hydronephrosis.

Pathology

Gross picture

The pelvi-calyceal system is transformed into a multilocular cavity filled with pus and lined with necrotic renal parenchymal tissue. There is marked perinephritis with dense adhesions and fibrofatty proliferation. Secondary calculi, with calculus pyonephrosis, are common.

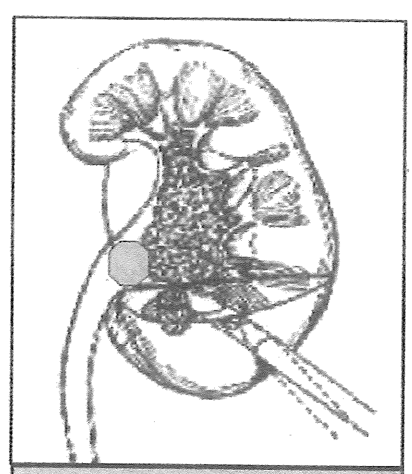


Fig. 45.3. Nephrostomy to drain an obstructed infected renal pelvis.

Complications

Septicemia and septic shock.
Renal scarring and damage.

Clinical features

1. **Pain**. Loin pain is worse at night, and has a constant aching character. Renal colic may occur with the passage of pus or stones down the ureter.
2. **Fever**. Evening rise of temperature to 37.5° or 38°C. Considerable rise may occur from retention of purulent urine.
3. **Swelling**. The kidney is palpably enlarged, tender and with limited mobility. Fluctuation cannot be elicited. The swelling is usually small, but when due to infection of a hydronephrosis, it may be very large.
4. **Pyuria**. Urine is turbid and contains large quantity of pus, which quickly settles to the bottom of a glass. Pyuria gives cystitis. Pus may be absent if infection is closed.

Investigations

1. Urine analysis and culture.
2. Abdominal sonography: shows the dilated kidney with pus inside
3. Radiography: A plain X-ray may reveal a calculus. Intravenous urography shows delayed or absent secretion of contrast media.
4. Ascending pyelography: is required in some cases to confirm the diagnosis and to drain the kidney.

Differential diagnosis is similar to that of acute pyelonephritis.

Treatment

1. An obstructed infected kidney should be treated urgently: Antibiotics and kidney drainage by inserting a large percutaneous nephrostomy tube (Fig. 45.3) or ureteric catheter. Any cause of obstruction as a stone or stricture should be corrected.
2. Nephrectomy: In advanced cases with atrophic parynchyma and total renal function loss as detected by radio-isotpic renal scan, primary nephrectomy, may be considered provided the opposite kidney is normal. Preliminary nephrostomy may be done first.

Perinephric abscess

A perinephric abscess is suppuration of perinephric fat and fascia (Fig. 45.4).

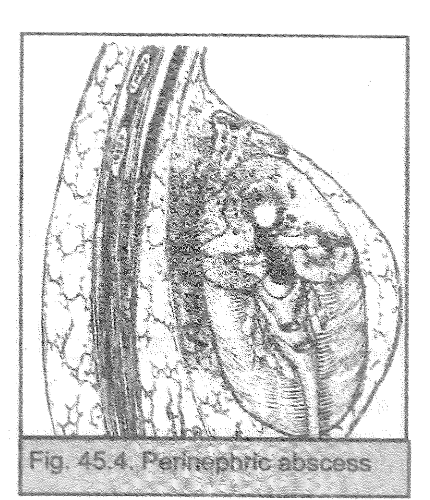


Fig. 45.4. Perinephric abscess

Aetiology

A perinephric abscess usually affects adults, more on the right side. It occurs in two forms:

1. Primary perinephric abscess is rare. The infection is blood-borne from a distant septic focus, such as a boil or carbuncle.
2. Secondary Perinephric abscess is more frequent due to extension of infection from the kidney or a neighboring structure, e.g. spine, pleura or pelvic organs.

Pathology

- The infecting organisms are staphylococci, less commonly streptococci and E. coli.
- Pus collects between the kidney and perinephric fascia and may extend downwards into the pelvis along the course of the ureter, or may burst through the perinephric fascia to reach the loin.
- The abscess may be unilocular or multilocular.
- A neglected abscess may be complicated by septicaemia and septic shock.

Clinical features

Symptoms

- Loin pain. Pain increases by movement, respiration and coughing.
- Hectic fever.

Signs

1. Fever
2. Loin fullness, tenderness and rigidity. Swelling is ill defined and does not move with respiration.
3. Oedema and redness of skin may be present if the abscess extends superficially.
4. Slight eversion and flexion of hip joint from spasm of psoas muscle.

Investigations

1. Urine analysis is usually sterile.
2. Blood picture shows polymorphonuclear leucocytosis.
3. Ultrasound or CT scan is diagnostic (pus around kidney).
4. Plain X-ray may show scoliosis with the concavity towards the abscess side, together with oblitration of the psoas shadow and elevation and fixation of the diaphragm.

Differential diagnosis is similar to that of acute pyelonephritis.

Treatment

- As for any abscess in the body treatment is by **drainage of pus**. Adequate lumb incision under general anaesthesia. All pockets are thoroughly opened. A wide drain Inserted.
- Antibiotics.

Cystitis

Aetiology

Organism. E. coli is the commonest organism and may occur in single or mix infections. Others include proteus, Streptococcus faecalis and Pseudomonas pyocyanea

Predisposing factors

1. Incomplete emptying of the bladder that leads to accumulation of residual urine, e. prostatic obstruction, urethral stricture, pregnancy and neurogenic bladder.
2. Constant reinfection from the upper or lower urinary tract, or from neighbouri structures such as the prostate, colon and cervix uteri (e.g., contraceptive device).
3. Lowered general resistance, e.g., diabetes.
4. Devitalization of the bladder by instrumentation, calculi, foreign bodies and growths.

Routes of infection

1. Ascending (commonest) from the urethra, prostate and vulva or duri instrumentation.
2. Descending from the kidney.
3. Lymphatic from the cervix uteri, prostate, seminal vesicles or bowel.
4. Direct through a vesicovaginal or vesicointestinal fistula.
5. Haematogenous from distant foci, e.g. boils, carbuncles and infected tonsils.

Clinical features

Symptoms

1. Increased frequency of micturition, both diurnal and nocturnal, is the chief symptom
2. Suprapubic and perineal pain. Severe pain is present at the end of micturition and referred to the glans penis or labia majora.
3. Pyuria. Urine is cloudy and a feathery deposit may appear on standing.
4. Haematuria. The last few drops of urine are blood-stained (terminal haematuria), a in severe cases the whole specimen may be blood-stained.
5. Pyrexia rarely occurs in acute cases and it seldom reaches 38°C.

Signs

- Tenderness over the bladder on abdominal, rectal or vaginal examination.
- Urethral discharge, tender prostate (prostatitis), chronic cervicitis or erosion may I detected.

Investigations

1. **Examination of the urine**. A mid-stream specimen sent for analysis with culture a antibiotic sensitivity tests. In acute stage, Instrumentations are contraindicated.
2. **Cystoscopy**. In chronic cases, it may reveal the precise pathological lesions, a local predisposing causes in the bladder, urethra or prostate e.g. bladder ulcer.

Treatment follows the previously mentioned general principles.

Gonococcal urethritis

Gonorrhea is a sexually-transmitted disease. Neisseria gonorrhea are Gram-negative diplococci located within the neutrophils. The urethra is a common site of infection. Concurrent infections with Chlamydia and other organisms are common.

Clinical features

Symptoms

- The usual incubation period is 3-10 days.
- May be asymptomatic
- In males there are urethral itching, urethral discharge and dysuria.
- Without treatment, urethritis will stay for 3-7 weeks. 95% of men become asymptomatic after 3 months.
- Complications such as involvement of the prostate may lead to urinary frequency, urgency, and nocturia. Spread down the yas deferens to the epididymis may lead to acute epididymitis.

Signs

- Urethral discharge is yellow or brown.
- There may be meatal oedema and erythema.

Investigations

- A gram-stained smear is the usual method of diagnosis. It is considered positive if Gram-negative diplococci are seen within polymorphonuclear leukocytes. Cultures are not necessary for diagnosis, but may help to determine antibiotic sensitivities.
- Alternative diagnostic methods are based on detection of the gonococcal enzymes, antigens, DNA, and liposaccharides.

Differential diagnosis

Non-gonococcal urethritis, the discharge is more scant and clear.

Complications

1. Periurethritis, abscess formation, urethral fibrosis, and, finally, urethral stricture.
2. Prostatitis may develop and may cause perineal pain and low backache.
3. Epididymitis may occur and could lead to infertility or testicular atrophy.
4. Tenosynovitis and arthritis may occur.

Prevention

Condoms, if properly used, will prevent the spread of N. gonorrhea.

Treatment

- Specific measures. Antibiotic treatment.
- Treatment of the sexual partner is imperative.
- General measures. Sexual intercourse should be avoided until cure has been established.

Non-gonococcal urethritis

Non-gonococcal Urethritis accounts for 50% of urethritis.

Etiology

- Organism. Chlamydia trachomatis is a small obligate intracellular bacterium of columnar or pseudocolumnar epithelium.

- Postgonococcal urethritis occurs in those who get gonorrhea and chlamydial infection simultaneously.

Clinical features

The disease usually presents after a 7-21 day incubation period, with dysuria and mild whitish or clear scanty urethral discharge. Discharge may be absent, and patient may only complain of urethral itching. Asymptomatic infection occurs in contacts of women with known cervical chlamydial infection.

Treatment of the patient and sexual partner is by Tetracycline.

Specific infection

Urinary Bilharziasis

Incidence

The disease affects adolescents and adults between the ages of 10 and 30. Males suffer more than females (4:1). The majority are those whose occupations entail exposure to water canals especially of small streams, such as farmers and fishermen. The parasite's life cycle is shown on Fig. 45.5.

Aetiology

The parasite Schistosoma haematobium is the usual cause (96%) but rarely (4%) Schistosoma mansoni is implicated. The worms migrate from the liver to the mesenteric veins, and reach the urinary bladder through the anastomotic channels between the haemorrhoidal and vesicoprostatic plexuses.

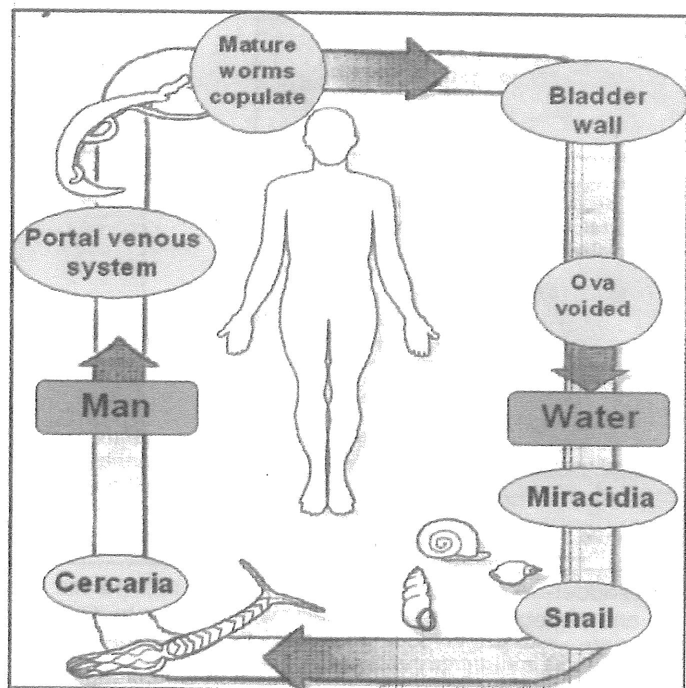


Fig. (45.5): Life cycle of Schistosoma worms

Pathology

The urinary bladder is the, commonest site of Bilharzial infection.

1. Patchy hyperaemia over the trigone.
2. Bilharzial sandy patches. Aggregations of calcified dead ova in the submucosa, which appear through the mucous membrane like sand under water. They tend to form a barrier to the passage of ova, so normal urine does not mean a cure of the disease.
3. Bilharzial tubercles. are larger, more numerous and more yellow than those of tuberculosis. They consist of small bilharziomata, each the size of a pin's head and surrounded with a narrow ring of congestion. They may undergo resolution, calcification or ulceration.
4. Bilharzial nodules, are spheroidal bodies, larger and more prominent than the tubercles. They are greyish in colour and may be discrete or aggregated. They

consist of crypt-like invaginations of hyperplastic epithelium, having a pseudoglandular or cystic appearance (cystitis glandularis and cystitis cystica).

5. Bilharzial papillomata. are polypoid projections of the mucous membrane due to irritation and hyperplasia of the epithelium. They vary in size from that of a pea to that of an almond. They vary in number and distribution, and lie commonly in the vicinity of the ureteric orifices, trigone and base of the bladder, and when the bladder is grossly infected they undergo necrosis, ulceration and encrustation with phosphates crystals. They may obstruct the flow of urine from the ureter or bladder but rarely become malignant.
6. Bilharzial granulomata. are submucous masses of bilharzial granulation tissue, which form smooth tumour-like swellings projecting into the bladder.
7. Bilharzial ulcers are usually single and small with clean-cut edges and superficial pale yellowish floors covered with scanty granulation tissue. They occur most frequently on the posterior wall of the bladder and are often surrounded by congestion and sandy patches. They tend to heal under treatment.

Complications

1. Secondary infection is very common. It may lead to any form of acute or chronic cystitis.
2. Stone formation from stasis, changing pH and deposition of debris and blood.
3. Ureteric obstruction from infiltration and fibrosis of the ureter. It usually affects the intramural portion and less commonly the juxtavesical portion. The stricture is usually bilateral and leads to progressive dilatation of the kidneys and ureters.
4. Bladder-neck obstruction due to fibrosis around the internal meatus. Obstruction leads to hypertrophy and trabeculation of the bladder and later on to dilatation and incomplete emptying (residual urine). The obstruction is aggravated by infiltration of the detrusor with atrophy of the muscle fibres and progressive fibrosis and loss of contractility.
5. Pericystitis from extension of the disease from the bladder, ureters, prostate and seminal vesicles. The ova are deposited in the loose pelvic cellular tissues, leading to the formation of a dense tough fibrous sheath, which causes progressive contraction and deformation of the bladder.

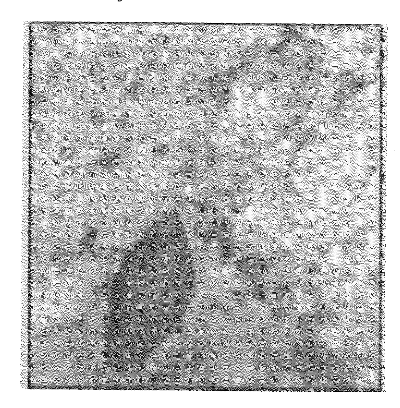


Fig. (45.6): S haematobium ova and RBCs in urine

6. Associated lesions: Spreads to seminal vesicles, prostate, urethra and scrotum with consequent urethral strictures and fistulae and multiple bilharzial masses and nodules.
7. Malignancy: Bilharzial cancer of the bladder is a common malignant tumour in Egypt. The malignancy commences most often in patches of leukoplakia or alkaline encrusting cystitis. Cystitis cystica, cystitis glandularis, ulcers and papillomata are rarely precancerous. The factor, which determines the onset of malignancy, is obscure. It is supposed that alkaline sepsis with ammoniacal decomposition is the most important factor. In favour of this theory, is the absence of such alkaline decomposition from the colon and its constant presence in association with bilharzial carcinoma of the bladder. It is known that bilharzial lesions of the colon are not premalignant.

Clinical features

Swimmer's or bather's itch due to penetration of cercaria through the skin may be followed 4-12 weeks later by fever, urticaria and asthma. Such symptoms usually pass unnoticed until ova invade the bladder within 6 months after cercarial penetration.

Symptoms

1. Haematuria is the cardinal symptom. It is usually slight (few drops), and terminal.
2. Pain. Some urethral burning may be felt, but with the occurrence of ulceration, stone formation or malignancy, the pain is markedly increased.
3. Frequency: From congestion of the trigone but later on severe frequency follows secondary cystitis and other complications.
4. Difficulty in micturition. At first there may be a slight difficulty in voiding, together with a feeling of discomfort and heaviness in the perineum at the end of the act. In late cases, severe difficulty may occur from bladder neck obstruction.

Signs

No signs are elicited until complications supervene.

Investigations

1. Urine examination. In the acute congestive stage, urine looks smoky and contains living bilharzia ova and red cells (Fig. 45.6). When secondary infection occurs, it becomes alkaline and turbid with blood, pus, phosphates, epithelial debris and dead ova.
2. Plain radiography shows Bilharzial calcification of bladder (Fig. 45.7), terminal ureters, seminal vesicles (honey coomb pattern), calcified papilloma or calculi.
3. IVU may show strictures of the ureters, hydronephrosis or bladder neck obstruction.
4. Cystoscopy. Cystitis, ulcers or tumor.

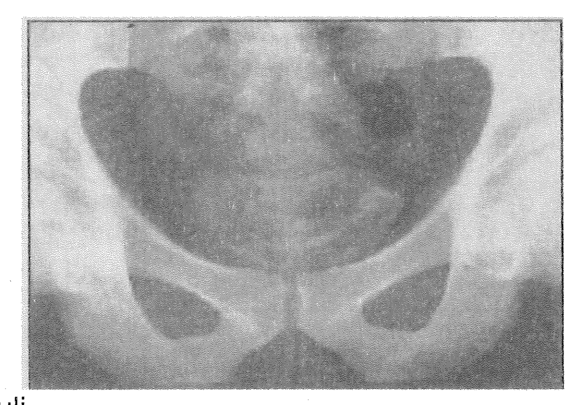


Fig. (45.7): Calcified Bilharzial bladder on plain X-ray

Treatment

1. Anti-Bilharzial drugs.
2. Urinary antiseptics should be given if secondary cystitis is present.
3. Surgical treatment depends on the individual lesion:
    a. Cystitis cystica, cystitis glandularis and Bilharzial papillomata require no special treatment unless they are bulky and causing obstruction. Cystoscopic fulguration or excision biopsy may be needed.
    b. Superficial bladder ulcers need cystoscopic excision. Partial cystectomy is necessary for intractable cases and for deep ulcers.
    c. Bladder neck obstruction may need cystoscopic incision or resection.
    d. Lower ureteric stricture may require uretroscopic balloon dilatation, or uretero-vesical implantation.

OBSTRUCTIVE UROPATHY

CHAPTER CONTENTS

General principles

Urinary tract obstruction, or obstructive uropathy, signifies any obstruction that occurs anywhere in the urinary tract. It is associated with changes in the urinary system above the level of obstruction.

Classification

- Acute or chronic obstruction.
- Congenital or acquired obstruction.
- Partial or complete obstruction.
- Extrinsic or intrinsic obstruction.
- Unilateral or bilateral obstruction.

Aetiology (according to the level of obstruction)

Renal causes

1. Kidney stones.
2. Congenital (idiopathic) pelvi-ureteric junction obstruction (PUJO).
3. Aberrent renal vessels (Fig. 43.7).
4. Renal pelvic tumors.
5. Tuberculous stricture.

Ureteric causes

1. In the lumen, e.g., stones and blood clots.
2. In the wall
   a. Congenital atresia and ureterocele.
   b. Ureteric stricture. Bilharzial, tuberculous or iatrogenic.
   c. Tumours.
3. Outside the lumen (compression)
   a. Infiltration by carcinoma of the cervix, colon, or rectum.
   b. Idiopathic retroperitoneal fibrosis.

Urinary bladder causes

1. Tumours involving uretero-vesical junction, whether primary or secondary.
2. Neurogenic bladder dysfunction.

Bladder neck causes

1. Congenital bladder neck obstruction.
2. Bilharzial bladder neck obstruction.
3. Bladder neck tumours from the urinary bladder or local extension from prostate or cervix.

Prostate causes

1. Benign prostatic hyperplasia (BPH).
2. Carcinoma of the prostate.

Urethral causes

1. Congenital causes as meatal stenosis and posterior urethral valves.
2. Stricture of the urethra.

Pathological changes

Accumulation of urine causes the following back pressure changes above the obstruction (Fig. 46.1). **Urethra.** Increased hydrostatic pressure leads to urethral dilatation.

Urinary bladder

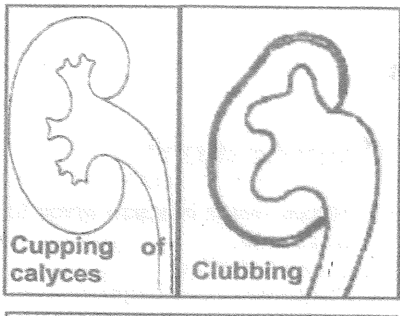
- **Early**, in the compensated phase, hypertrophy of muscle layer of the bladder occurs to counteract the increased urethral resistance. The bladder wall becomes trabeculated. Saccules form when the mucosa protrudes between muscle fibres. Diverticulae develop when mucosa bulges outside bladder wall.
- **Later**, with unrelieved obstruction the bladder dilates and its wall thins out. Atony of the bladder develops and leads to accumulation of residual urine with subsequent retention of urine.

Ureter.

Early there is muscular hypertrophy of the wall, later atony occurs. The ureter becomes dilated (hydroureter), elongated and tortuous.

Kidney

- Morphological changes. Normally, the pressure within the renal pelvis is close to zero. With obstruction this pressure increases and pelvi-calyceal system dilates leading to hydronephrosis.
  - In early stages, renal pelvic musculature undergoes compensatory hypertrophy to force urine past the obstruction. Later, muscles become stretched and atonic (decompensated).
  - Calyces are the first to suffer from obstruction. The normal concave appearance (cupping) of minor calyces , as it appears on a urogram, becomes flat then rounded (clubbing) and lastly ballooned.
  - Renal parenchyma starts to thin out as it suffers from ischaemic atrophy as it is compressed between renal capsule externally, and the centrally increasing intra-pelvic pressure.
  - At a later stage, the kidney is completely destroyed and appears as a thin-walled sac filled with clear fluid or, if infected with pus.
- **Functional changes**
  - Intra-pelvic pressure continues to rise till it overcomes the glomerular filtration pressure, where urine secretion stops.



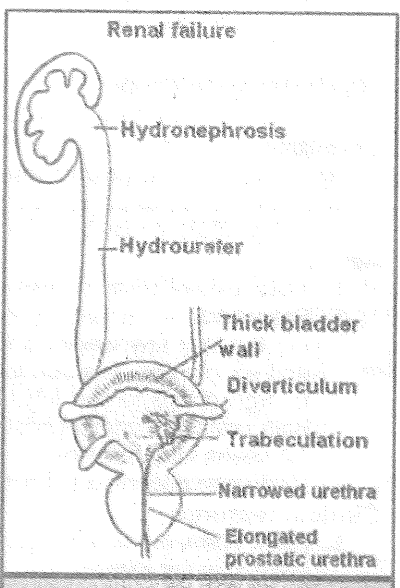


Fig. 46.1. Summary of the pathological changes that occur proximal to urinary obstruction. In this case the cause is BPH.

- Acute obstructive conditions that need urgent interventions are:
  1. Acute retention of urine (see Chapter 42).
  2. Calcular anuria.
  3. Ligation of both ureters during a surgical procedure (see Chapter 44).
- Bilateral obstructions or infravesical obstructions are serious because they affect both sides of urinary tract with resulting uraemia.
- Obstructive uropathy consequences are reversible when treated early.

- Pyelo-venous, pyelo-lymphatic, and pyelo-tubular back flow occur. Urine leaking into the interstitial tissues is absorbed by the renal lymphatics.
- Glomerular filtration rate and the renal plasma flow are reduced.
- Renal concentrating power is gradually lost.
- The contralateral normal kidney undergoes compensatory hypertrophy in order to maintain an overall normal renal function.
- In neglected bilateral obstruction, renal failure will occur.

A combination of urinary obstruction and infection leads to rapid deterioration of kidney function.

With relief of obstruction

- Early treatment leads to remarkable improvement of morphology and function.
- Delay in treatment results in some degree of irreversible renal damage.

Factors affecting morphological and functional changes following obstruction

1. Level of obstruction. Obstructive lesion at or below the bladder neck will have a bilateral effect, while lesion in the ureter or pelvi-ureteric junction will affect one kidney only.
2. Degree of obstruction. Severe obstruction leads to more damage to the kidney.
3. Duration of obstruction. Long-standing partial or intermittent obstruction tends to produce significant proximal dilatation. Acute obstruction, on the other hand, tends to produce mild dilatation but more rapid deterioration of function.
4. Development of infection on top of obstruction causes very serious deterioration of function of the affected kidney.



Fig. 46.2. Hydronephrosis caused by a stone in the pelvis.

Complications

1. Stasis invites infection which further deteriorates kidney function at a fast rate.
2. Stone formation.
3. Rupture of hydronephrotic kidney (rare, with trauma).
4. Renal insufficiency and ultimately failure in bilateral cases.

Fig. 46.3. IVU showing right hydronephrosis that is caused by BPH.

Hydronephrosis

Definition

Hydronephrosis is chronic aseptic distension of the renal pelvis and calyces due to partial or intermittent obstruction of the urinary tract (Fig. 46.2). It may be unilateral or bilateral.

Aetiology

1. All causes of chronic obstructive uropathy mentioned before.

2. Pregnancy; especially on the right side. Dilatation occurs during the first few weeks and reaches maximum between fifth and six months. It is due to atony of ureteric musculature due to high progesterone level. Also, the foetal head may press both ureters. Hydronephrosis of pregnancy subsides within 2- 12 weeks after delivery.
3. Vesicoureteric reflux (Chapter 45).

Clinical features

1. Pain. There is usually slight dull aching pain or a sense of discomfort in the loin.
2. Swelling. In late stages a cystic swelling filling the renal angle can be palpated.
3. Polyuria of low specific gravity.
4. Manifestations of renal failure, if present.

Complications. See general principles of obstruction.

Differential diagnosis of palpable hydronephrosis

1. Polycystic kidney.
2. Hypernephroma.

Investigations

1. Urine analysis for RBCs, WBCs, SG and crystals.
2. Ultrasound examination to detect renal size, thickness of the renal cortex and possibly the cause, e.g., stones.
3. IVU (Fig. 46.3) will show the following:
    a. Dilatation of renal pelvis which becomes biconvex.
    b. Pelviureteric junction is distorted and is raised upwards.
    c. Loss of cupping, clubbing and later ballooning of the minor calyces. The major calyces are also dilated.
    d. Delayed excretion of contrast. The secretion is delayed and the concentration of the medium is below normal. In advanced cases no secretion occurs.
4. Ascending (retrograde) pyelography (Fig. 42.5). If IVU. does not give conclusive data, ascending pyelography is indicated to confirm the diagnosis and to see the cause.
5. Descending (antegrade) pyelography is sometimes done if a nephrostomy tube is present.

Treatment

- Removing cause of obstruction is usually followed by recovery of the kidney.
- Nephrectomy is avoided as far as possible- Only when all the following criteria are present nephrectomy is justified.
    - The kidney is non functioning with infusion urography.
    - Sonography showed very thin parynchyma.
    - Renal scan shows zero function.
    - The opposite kidney is normal.
- In compromised cases with bad other kidney, a percutaneous nephrostomy (Fig. 45.3) may be left for long time till obstruction is treated.
- In advanced cases of pelvic hydronephrosis a plastic operation is performed to reduce the size of the pelvis and to provide adequate drainage of the lowest part of the pelvis (Fig. 43.9).

Ureteric stricture

This is narrowing of the ureter that impedes urine flow.

Aetiology

1. Congenital. At pelviureteric junction obstruction (PUJO) or at ureterovesical junction (pin-hole meatus).
2. Traumatic. Follow open or closed injuries of the ureter particularly operative or instrumental injuries.
3. Inflammatory
   a. Bilharzial stricture commonly affects the intramural and lower part of the ureter and less commonly opposite the third lumbar vertebra. It may be bilateral.
   b. Tuberculus stricture affects the whole ureter and is usually multiple and bilateral.
4. Long-standing stone may lead to stricture.
5. Neoplastic. Carcinoma of the ureter or more often, invasion of ureter by a malignant growth as carcinoma of the uterus, colon, or rectum.

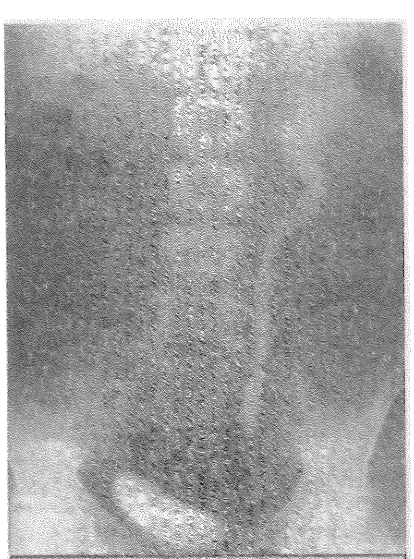


Fig. 46.4. IVU showing iatrogenic stricture of left ureter following hysterectomy. This is a delayed film where the Rt kidney has already excreted the contrast. On the Lt side there is hydroureter and hydronephrosis with delayed excretion and emptying of contrast.

Clinical features

1. Renal aching pain due to distension of the renal pelvis.
2. Progressive hydronephrosis.
3. Recurrent attacks of pyelonephritis.
4. In Egypt, bilateral hydronephrosis is a common sequel of bilateral Bilharzial ureteric stricture. These patients may present by chronic renal failure.

Investigations

1. Blood urea, serum creatinine and serum electrolytes.
2. Sonography will demonstrate hydronephrosis.
3. Intravenous urography will determine site of the stricture and degree of functional capacity of the two kidneys (Fig. 46.4).
4. Retrograde and antegrade pyelography (see hydronephrosis).



Fig. 46.5. Uretero-vesical implantation.

Treatment (mainly surgical)

1. Stricture of upper two thirds of ureter (above the level of ischial spine). Excision and end-to-end anastomosis is done for localized strictures. Both ends of the ureter are trimmed obliquely, spatulated and then anastomosed by interrupted sutures of 4-0 Vicryl over a ureteric splint which is removed later by a cystoscopic forceps.
2. **Stricture of lower third of ureter**
   a. Uretero-vesical implantation is done for localized stricture involving the lower end of the ureter as commonly occurs in Bilharziasis. The ureter is divided above the stricture and then a new ureterovesical anastomosis is established. The ureter is passed in a submucous tunnel in the bladder before anastomosis is performed to make a valve (Fig. 46.5) in order to prevent vesico-ureteric reflux.
   b. Bladder (Boari's) flap is done for a long stricture of lower part of ureter when direct ureterovesical anastomosis is not feasible. A flap can be prepared

from wall of the bladder, transformed to a tube and anastomosed to ureter (Fig. 46.6).

3. **Stricture of long segment of ureter**. Ileal loop replacement. The ureter can be replaced by a segment of ileum that retains its blood supply. One side is anastomosed to renal pelvis and the other side to the bladder.

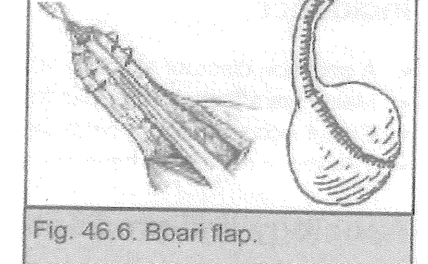
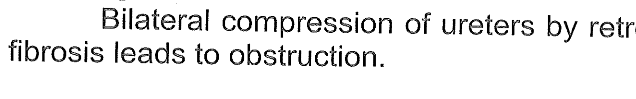


Fig. 46.6. Boari flap.

Retroperitoneal fibrosis



Bilateral compression of ureters by retroperitoneal fibrosis leads to obstruction.

Types

Primary (idiopathic) retroperitoneal fibrosis

- Unknown aetiology but may be drug-induced, e.g., methysergide that is used for prophylaxis against migraine.
- The patient may present acutely with anuria. Ureteric catheterization is urgently done to relieve obstruction. Unlike calcular anuria, the catheters pass up to the kidneys easily.
- ESR, urea and creatinine are usually elevated.
- IVU and/or ascending pyelography demonstrates that both ureters are pulled medially towards the midline.
- CT shows a retroperitoneal fibrous plaque in which both ureters and great abdominal vessels (aorta and IVC) are embedded. Both kidneys show hydronephrotic changes.
- Definitive treatment is surgical ureterolysis, i.e., release of ureters from fibrous tissue, then wrapping them in pedicled omentum inside the peritoneal cavity to avoid re-obstruction. Postoperative corticosteroid therapy is believed to reduce the possibility of recurrence.

Common causes of stricture of the ureter include Bilharziasis that affects the lower ureter mainly and TB that affects the whole ureter and is multiple. Treatment of ureteric stricture is essentially surgical.

Secondary retroperitoneal fibrosis

- Diffuse infiltrating retroperitoneal malignancy.
- Previous leakage of an aortic aneurysm.

Urinary stones

Incidence

- A common disorder affecting 10-20% of the population at some stage of their life.
- Males are affected more than females.
- Calculi occur mainly in middle aged persons but no age is exempt.
- Two thirds of patients have one or more recurrences within the following 8 years.

Aetiology and predisposing factors

Inadequate drainage

Normal urine flow expels crystals before they have the chance to coalesce and grow. Stasis, e.g., BPH and strictures allow stones to form. Stasis also predisposes to infection.

Excess normal constituents in urine

A high concentration of solutes in urine results in precipitation of crystals, which initiates stone formation.

- **Inadequate** urine volume. Urinary stones are prevalent among people who live in warm and hot climates where dehydration produces extremely concentrated urine.
- **Excess urinary excretion of calcium**. The causes may be:
  - Idiopathic
  - Hyperparathyroidism.
  - Prolonged immobilization.

Fig. 46.7. Plain X-ray showing a staghorn stone in right kidney.

- **Excess urinary excretion of uric acid**. The causes may be:
  - Gout. Purine-rich food, e.g., meat, liver and kidney, increase uric acid level in urine.
  - Chemotherapy for leukaemias and lymphomas.
- **Excess urinary excretion of oxalates**. The causes may be:
  - Idiopathic hyperoxalluria.
  - Excess dietary intake of strawberries, green leafy vegetable and tea.
  - Loss of terminal ileum, e.g., by resection or Crohn's disease.

Fig. 46.8. Complications of renal and ureteric calculi.

Presence of abnormal constituents in urine

- **Infection**
  - Infection produces epithelial desquamation upon which calculi may deposit.
  - Infection with urea-splitting organisms cause alkalinization of urine and favours formation of phosphate stones.
- **Foreign bodies** such as non-absorbable sutures placed at operation, ureteric stents or fragments of broken catheters may act as a nidus for stone formation.
- **Vitamin A deficiency** is rare but cause hyperkeratosis of urothelium. The epithelial debris also acts as nidus for stone formation.
- **Cystinuria** is an inborn error of metabolism, which results in excess cystine excretion in urine, and subsequent cystine some formation.

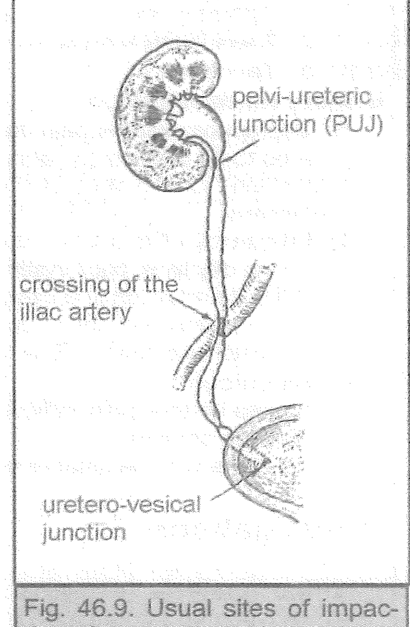


Fig. 46.9. Usual sites of impaction of a ureteric stone.

Composition of urinary calculi

- **Calcium calculi** are either calcium oxalate, calcium phosphate or a mixture of the two. Together they form the majority of urinary stones (75%).
  - Calcium oxalate calculi are the most common type and are radio-opaque. They are hard and have spiky surfaces, which injure the epithelium during their movement. This causes bleeding, which accounts for their dark brown or black colour.

- **Calcium phosphate** stones are smooth, yellowish white and radio-opaque. They grow in alkaline medium and can attain a large size, e.g., staghorn calculus that fills renal pelvis and calyces (Fig. 46.7).
- **Ammonium magnesium phosphate (struvite)** calculi (15%) form in foul decomposing alkaline urine and grow very rapidly, often filling the renal pelvis and calyces. They are friable, amorphous and have a smooth surface and a yellowish white colour. They are radio-opaque.
- **Uric acid calculi** (7-9%) are hard, smooth and golden yellow. Pure uric acid calculi are radiolucent, and will not be seen on plain films. Excretory urograms demonstrate a filling defect in the collecting system. They are demonstrated by ultrasound or CT.
- Cystine stones (1-3%).
- Xanthine stones are very rare.

Complications (see urinary obstruction, Fig. 46.8)

1. Obstruction
   a. Ureteric stones produce hydroureter and hydronephrosis.
   b. Stone impacted in pelvi-ureteric junction causes hydronephrosis.
   c. Stone impacted at neck of calyx causes dilatation of this calyx (hydrocalyx).
2. Infection. Urinary stasis encourages infection. In addition, some stones, as the struvite variety, are formed in an already infected urine.
3. Haematuria.
4. Deterioration of renal function. Either obstruction or infection affects the renal function. A combination of both seriously and rapidly damages the kidney. If this is the only functioning kidney, renal failure will develop.
5. Metaplasia of urinary epithelium. Chronic irritation of transitional epithelium caused by friction by stone, either in renal pelvis or urinary bladder causes, squamous metaplasia. Further irritation is thought to lead to squamous cell carcinoma.

Clinical features

Renal and ureteric calculi

Symptoms

- Asymptomatic in some cases, e.g., staghorn stone or a tiny stone in a calyx.
- Symptoms occur when stones move along the renal pelvis and the ureter causing distension of the proximal part of urinary tract. Common sites for stone impaction are pelvi-ureteric junction, pelvic brim as ureter crosses iliac artery, and uretero-vesical junction (Fig. 46.9).
  - Pain is the main symptom (Fig. 42.1).
    - Fixed dull aching pain in renal angle from renal pelvic stones. It may radiate to the back posteriorly or to the hypochondrium anterioly. Pain increases by movement.
    - Ureteric colic occurs as the stone moves along the ureter. A stone in upper third causes colicky pain in the loin, which is referred to groin or testicles (males) or labia majora (females).
    - Iliac pain. When the stone moves to middle third, iliac pain is felt and may simulate appendicitis.
    - Pain at end of micturition and referred to the tip of penis is caused by a stone impacted in the lower end of ureter, usually associated with frequency and strangury.
  - Nausea and/or vomiting. During an attack of renal pain or ureteric colic, due to irritation of coeliac ganglion. Sometimes abdominal distension may be present.

- Haematuria may occur with an attack of ureteric colic. It may be gross or microscopic and is caused by mucosal injury during stone migration.

Physical signs

1. Patient is restless because of inability to obtain relief irrespective of position.
2. Fever is rarely present unless there is urinary tract infection.
3. Tenderness in the loin, and probably over the site of the stone.

Urinary bladder calculi

1. Suprapubic bladder pain due to contraction of bladder on the stone at the end of micturition. It is refered to tip of penis (or labia majora), or to the perineum. It is more frequent at day time (during micturitiion). During sleep or in recumbent position, the stone moves away from the trigone and the pain lessens.
2. Frequency of micturition due to irritation of bladder mucosa and trigone. At first frequency is only during day time, but if cystitis develops, it becomes diurnal (day) and nocturnal (night).
3. Difficult micturition and interruption of stream during micturition.
4. Acute retention of urine due to obstruction of bladder neck by the stone.
5. Terminal haematuria. Few drops may pass by the end of micturition from abrasions of the trigone.

Urethral palculi

1. Severe urethral pain, difficulty and interruption of micturtion stream.
2. Retention of urine.
3. If a stone is in the anterior urethra it can be palpated.

Investigations

1. **Urine analysis**. Microscopic or gross haematuria is present in at least 90% of patients. Crystals of same type that are forming the stone may also be demonstrated.

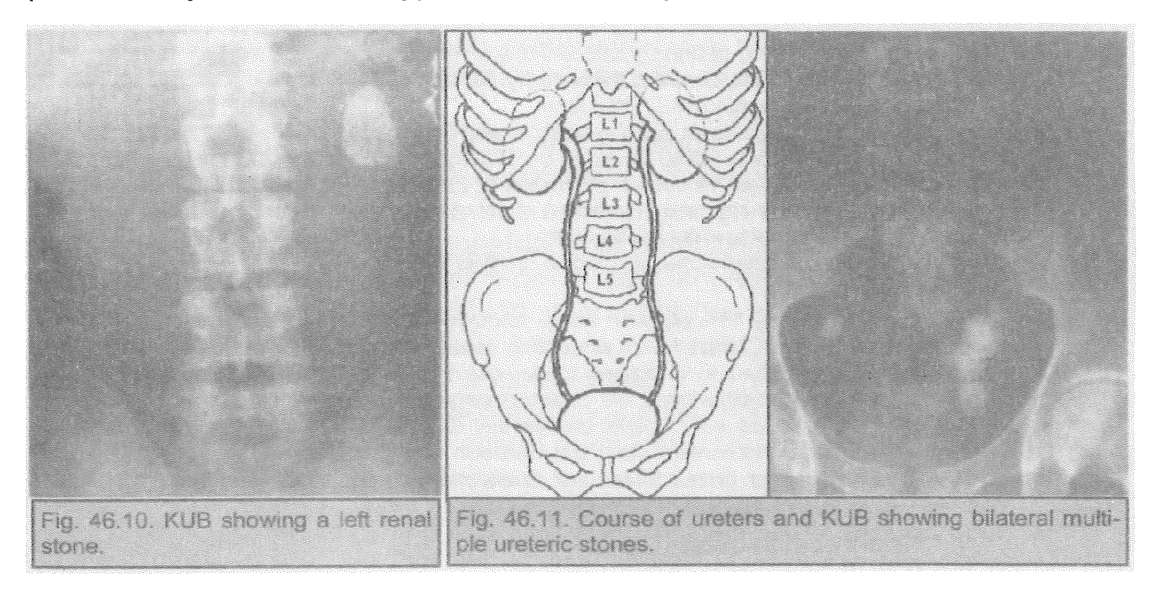


Fig. 46.10. KUB showing a left renal stone.

Fig. 46.11. Course of ureters and KUB showing bilateral multiple ureteric stones.

2. **KUB** (Fig. 46.7,10,11,12). 90% of urinary calculi show on plain x-ray. In descending order of radiodensity calcium phosphate is the densest, followed by calcium oxalate, struvite, cystine, and last of all is uric acid stone which is considered radiolucent.

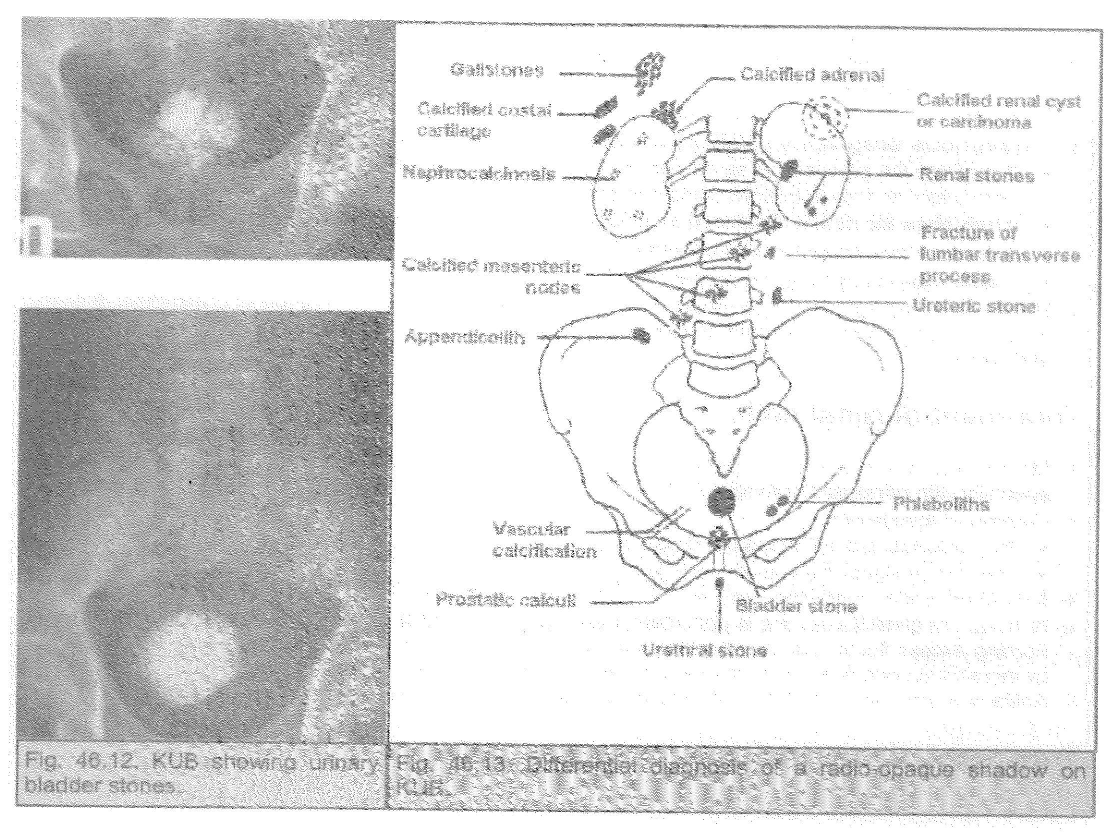


Fig. 46.12. KUB showing urinary bladder stones.

Fig. 46.13. Differential diagnosis of a radio-opaque shadow on KUB.

a. In order to suspect or to exclude an opaque shadow as a ureter stone it is importnt to remember the course of the ureter. It starts opposite the tip of transverse process of L1 (or L2) vertebra and then passes down along the tips of transverse processes of the following lumbar vertebrae. The ureter then crosses the pelvic brim opposite the sacro-iliac joint to enter the true pelvis. At the level of the ischial spine, it turns medially to enter the bladder.
b. **Differential diagnoses of a radio-opaque shadow on KUB** (Fig. 46.13)
   i. Urinary stones.
   ii. Gall bladder stones. Only 15% of these stones contain enough calcium to show on plain X-ray. They are usually multiple, faceted, with signet ring appearance and are present on the right side. Lateral view film shows them to lie anterior to the bodies of vertebrae near the anterior abdominal wall. A renal stone, on the other hand, will be seen overlying the vertebral body, posteriorly.
   iii. Pelvic phlebolith is calcified wall of pelvic vessels that simulate appearance of ureteric stones but commonly have radiolucent centers. On IVU a phlebolith is seen outside the ureter.
   iv. Calcified mesenteric lymph nodes. These are usually multiple, mottled and take the line of root of mesentry, i.e., from right iliac fossa to left upper abdomen.
   v. Nephrocalcinosis.
   vi. Calcified renal tumours.
   vii. Fracture tip of transverse process of a lumbar vertebra.
   viii. Calcified costal cartilage.
   ix. Calcifications in adrenal gland.
   x. Appendicolith and foreign bodies in the intestine.

3. **Intravenous urography (IVU)** is mandatory in all cases of urinary stones.

a. It confirms the presence of stone and determines its position. It is also useful in detection of radiolucent calculi as they appear as filling defects.
b. It may show the cause of stone, e.g., stricture or BPH.
c. It gives an idea about function of both kidneys and back-pressure effects, e.g., hydronephrosis.
d. It also helps in choosing the proper line of trearment for a paricular stone.

4. **Ultrasonography** is a simple means of visualizing the kidney and the degree of obstruction. It is also quite helpful to diagnose bladder stones and radiolucent calculi. It is not a reliable tool tO visualize ureteric calculi.

Treatment of renal colic

1. Most cases, are treated as outpatients. Hospitalization may be required for patients with severe colic specially with persistant vomiting.
2. Parenteral analgesics
   a. Non-steroidal anti-inflammatory drugs, e.g., indomethacin or diclofenac.
   b. Narcotic analgesics as opiates are given in resistant cases.
3. Anti-emetics, e.g., metochlopramide.
4. IV fluids are given if vomiting is persistent. Fluids are given according to normal patient's requirements. Forcing excess fluids in acute attack of colic would help flush out the stone but may also increase pain by increasing urine flow on an obstructed kidney.
5. Antibiotics are given only if there is suspicion of urinary tract infection.

Treatment of renal calculi

Conservative medical treatment

Prerequisites

1. Small stone less than 0.5 cm as 90% of them can pass spontaneously.
2. No evidence of back pressure effect (hydronephrosis).
3. No distal obstruction.
4. No evidence of infection.

Methods

1. High fluid intake with or without diuretics, e.g., frusimide to flush down the stone.
2. Analgesics and antispasmodics.
3. Urinary antiseptics.
4. Regular follow-up to monitor stone descent.
5. Recovering any stone or gravel that is passed either by filtering urine or by voiding in a clean container. Stone analysis allows planning for future therapy and prophylaxis.

Extracorporeal shock wave lithotripsy (ESWL)

Principle

This method depends on high-energy, electric-generated shock waves that are focused from outside the body on the stone after its visualisation using X-ray or ultrasonic imaging. The stone is fragmented to minute particles which will pass through urinary tract. High fluid intake is maintained to facilitate passage of gravels. The procedure is performed without anaesthesia, except in children.

Indications

All radio-opaque renal stones that are less than 2 cm whether calyceal or pelvic.

Advantages

1. Painless, non invasive technique which can be done as an outpatient procedure.
2. Suitable for risky patients.
3. Successful for most radio-opaque urinary calculi. Uric acid calculi are radiolucent and may cause some difficulty.

Contraindications

1. Urological contraindications:
   - Presence of distal obstruction (fragments will not pass).
   - Large stone (>2cm) except after debulking of stone by PCNL.
   - Renal insufficiency (kidney has no power to push fragments).
   - Acute episode of renal infection.
   - Stone in a solitary kidney (if required, stenting of ureter by catheter is mandatory to avoid acute obstruction by fragments which may cause acute renal failure).
2. Non urological contraindications
   - Pregnancy (X-ray exposure).
   - Bleeding diathesis.

Complications

1. Transient attacks of haematuria due to passage of gravel through the ureter.
2. Colicky ureteric pain during passage of fragments.
3. Ureteric obstruction from arrest of fragments in the ureter.
4. Fever due to acute obstruction by the fragments.
5. Failure to disintegrate hard stones.

Percutaneous nephrolithotomy (PCNL)

PCNL is an endoscopic technique to fragment and remove renal stones.

Indications

1. Large stones >2cm.
2. Cystine stones because they are very hard for ESWL.
3. Urinary obstructive lesions.
4. ESWL failure.

Contraindications

1. Bleeding diathesis.
2. Congenital anomalies e.g. horseshbe or ectopic kidney (has abnormal vessels).
3. During pregnancy (X-ray).

Technique (Fig. 46.14)

1. Access to the pelvi-calyceal system of the kidney. Contrast material is injected up a ureteric catheter to visualize the pelvi-calyceal system. A needle is passed to the pelvis through which a guide-wire is inserted. The tract is dilated over the guide wire to a size sufficient to accommodate the nephroscope.
2. Stone manipulation. Small stones (<1cm), can be directly extracted through the sheath. Large stones are fragmented first. Fragmentation through nephroscope can be done by either ultrasonic, laser or electrohydraulic lithotripsy.
3. Kidney drainage. After stone removal, a nephrostomy tube is left in situ for 48 hours to allow drainage of residual tiny fragments, urine and blood.

Advantages

1. Very small endoscopic incisions (1cm).
2. Minimal operative and post-operative side effects.
3. Short hospital stay (3 days).

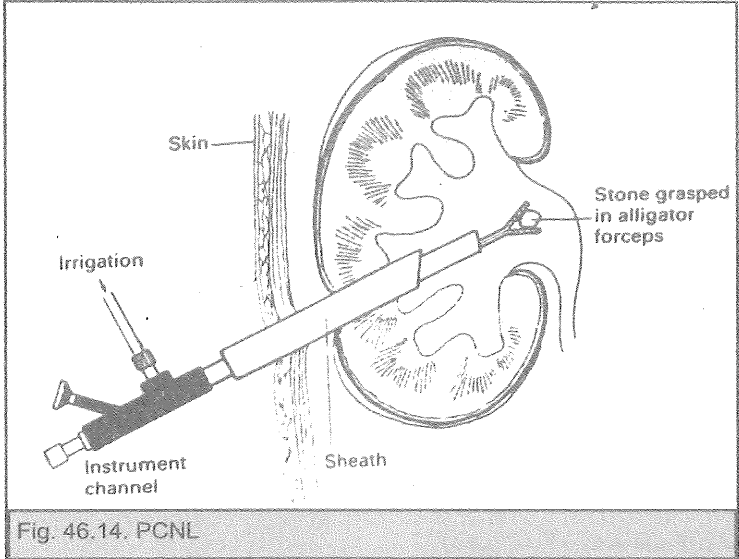


Fig. 46.14. PCNL

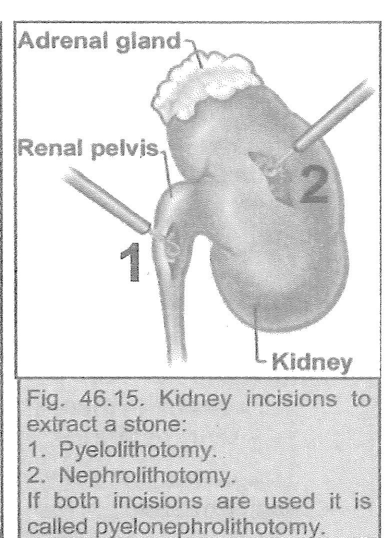


Fig. 46.15. Kidney incisions to extract a stone:
1. Pyelolithotomy.
2. Nephrolithotomy.
If both incisions are used it is called pyelonephrolithotomy.

Complications

1. Kidney bleeding.
2. Extravasation of the used irrigating fluid into peritoneal cavity, retroperitoneum or intravascular spaces.
3. Residual stone fragments.
4. Injury to adjacent organs, e.g., the colon.

Open surgery

Open surgery is the third choice for treatment for renal stones. Only 5-10% of patients with renal stones will be candidates for open surgery.

Indications

1. Contraindication to ESWL or PCNL.
2. Failed PCNL or ESWL.

Methods (Fig. 46.15)

1. Pyelolithotomy means opening the renal pelvis and removal of the stone. It does not cause any damage to renal parenchyma and bleeding is minimal. The renal pelvis is reached by exposing the posterior surface of the kidney as it is the most posterior structure.
2. Nephrolithotomy means incision through renal parenchyma to remove a stone. Nephrolithotomy is indicated when there is difficulty in exposing the renal pelvis or when stone is large and palpable through thin renal parynchyma.
3. Pyelonephrolithotomy combines the above-mentioned two procedures. It is indicated for branched calculi.
4. Partial nephrectomy is indicated for a stone impacted in a hugly dilated non function lower calyx with narrowing of its neck.
5. Nephrectomy is indicated if the kidney is non functioning provided that the contralateral kidney possesses a good function.

Surgical access to the kidney (Fig. 46.16)

1. A plain x-ray is done on the day of operation in order to confirm position of the stone.
2. Under general anaesthesia the patient is turned on his side (lateral position), with diseased side up and the bridge in the middle of the table is elevated to improve access.
3. The kidney is exposed by an oblique lumbar incision of Morris. This is a muscle cuffing incision that starts at the renal angle and is carried downward and forwards parallel to and about one inch below the last rib.
4. In the posterior part of the incision the latissmus dorsi and serratus posterior inferior muscles are divided. Anteriorly the two oblique muscles and the transversus abdominis are divided. The lumbar fascia is then incised and the peritoneum is peeled forwards. Incision of the perinephric fascia exposes the kidney.
5. The stone is removed by incising renal pelvis, parenchyma, or both as mentioned earlier.
6. Incision in the pelvis is closed by 3-0 and the kidney parynchyma by 2-0 polygalactin (Vicryl).
7. The wound is then closed in layers leaving a drain in the perinephric space.
8. The drain is removed after a few days when it stops to bring out urine. Skin sutures are removed on the 10th day.

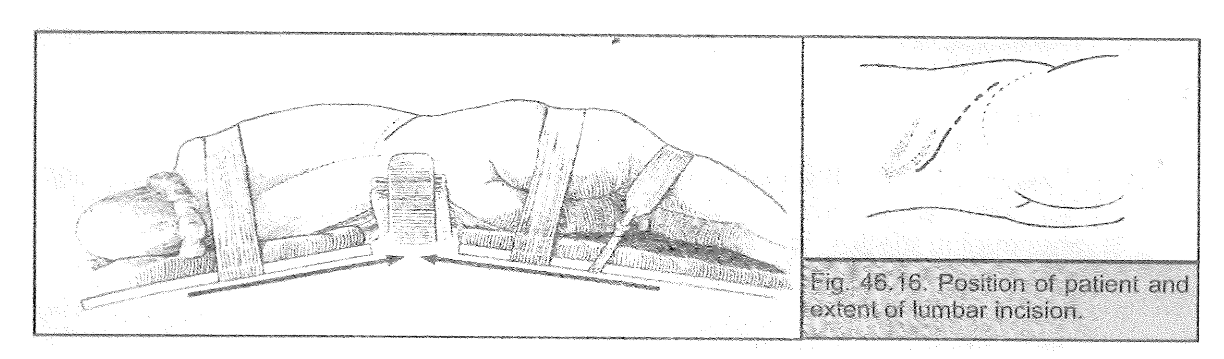


Fig. 46.16. Position of patient and extent of lumbar incision.

Treatment of ureteric calculi

Conservative treatment follows the same principles as for renal calculi.

Indications for intervention

1. Stones larger than 5mm.
2. Evidence of back pressure effect.
3. Evidence of secondary infection.
4. Persistent pain.
5. Failure of conservative treatment.

Types of intervention

Procedures for removal of ureteric calculi differ according to site of the stone and its size.

- **Upper third of ureter**
  - Stone less than 1 cm. ESWL with stone in place, or stone is pushed up to the kidney by a ureteric catheter and then ESWL is used as in renal stone.
  - Stone more than 1 cm. Open surgery is required.

- **Middle third of ureter**. Open surgery is the treatment of choice. Ureteroscopic extraction at this site is dangerous and not advised, ESWL is very difficult and not efficient.
- **Lower third of ureter**
  - Stones less than 1 cm can be extracted by ureteroscopy (URS).
  - Stones less than 2 cm can be fragmented and extracted by ureteroscopy.
  - Stones larger than 2 cm, open surgery is required.

Technique of ureteroscopy (URS)
- Under general, spinal or epidural anaesthesia, the patient is placed in lithotomy position on an operating table with radiological facilities.
- Cystoscopy to identify ureteric orifice.
- Fine ureteroscope is passed up the ureter under vision. The stone is visualized and if it is small, a wire basket is passed through the uretoerscope and is removed under vision.
- A large stone requires fragmentation by ultrasound, laser, or electrohydraulic lithotripsy under vision.

Complications of ureteroscopy (URS) are uncommon and include perforation, avulsion, or stricture of the ureter and infection.

Open surgery (ureterolithotomy)
Indications
1. Stones >1cm in upper ureter.
2. Stones >2cm in lower ureter.
3. Stones in middle ureter.
4. Associated ureteric stricture.
5. Failed URS or ESWL.
6. Contraindication to URS or ESWL.

Open surgery incisions and steps

The surgical access depends on the site of the stone.
- **Lumbar incision** for stones in upper third of the ureter. It is similar to that for exposure of the kidney.
- **Abernathy's incision** for stones in middle third of the ureter, as well as its lower third above the level of the ischial spine. It is a muscle cutting incision that starts above the anterior superior iliac spine and passes downwards and medially parallel to the lateral half of inguinal ligament.
- **Lower midline or Pfannenstiel's (transverse)** incision for stones in lower part of the ureter that lie below the level of the ischial spine.
  - As for the kidney a plain x-ray just before the operation is important.
  - At any level, the ureter is exposed by an extraperitioneal route, i.e., peritoneum is not opened but is peeled medially.
  - The ureter is identified by the following criteria:
    - Adherent to posterior peritoneum but easily peeled from it.
    - Longitudinal tubular structure.
    - Longitudinally arranged blood vessels.
    - Crosses the bifurcation of the common iliac artery.
    - Regular peristaltic contractions.
    - Stone may be felt inside it.
  - Once the ureter is identified a tape is passed around it proximal to the stone, otherwise the stone may slip upwards.

- It is preferable to open ureter in a healthy area above the stone.
- The stone is removed by a uroterolithotomy forceps.
- A small ureteric catheter (6F) is passed downwards the ureter to check its patency and to exclude any stricture.
- The ureter incision is closed by fine 4-0 polygalactin (Vicryl) suture.
- A drain is inserted and the wound is closed in layers.
- The drain is removed after a few days when it stops bringing out urine.
- Skin sutures are removed on the 10th postoperative day.

Treatment of urinary bladder stones

Due to high pressure in bladder during voiding, stones less than 1cm have no chance to stay in the bladder unless there is distal obstruction, e.g., stricture or BPH. For larger stones urgent intervention is required as urine retention may occur.

Cystoscopic litholapaxy

Indications

Stones less than 2 cm.

Method (Fig. 46.17)

1. The stone is crushed cystoscopically either mechanically by lithotrite (technique is called litholapaxy) or by ultrasonic waves or electrohydraulic shock waves.
2. Fragments are then lavaged cystoscopically to outside the bladder by Ellik's evacuater.

Complications include bleeding, injury to the bladder or urethra, and failure to crush the stone.

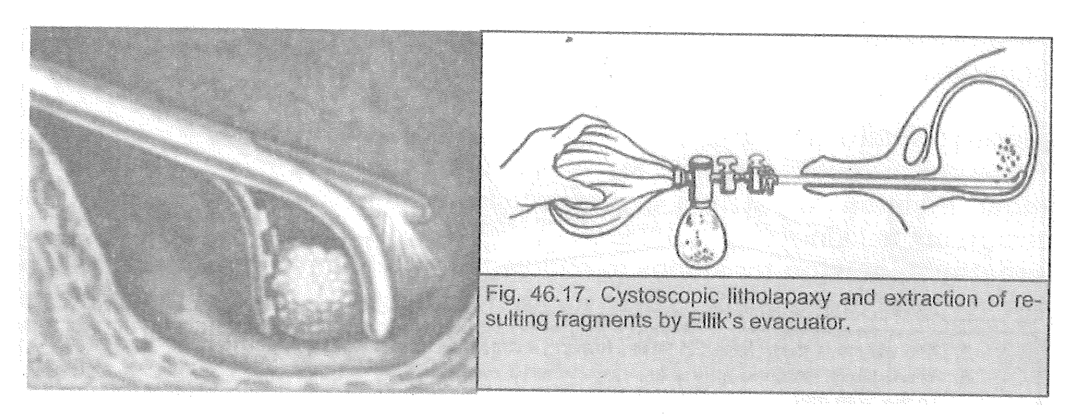


Fig. 46.17. Cystoscopic litholapaxy and extraction of resulting fragments by Ellik's evacuator.

Open surgery (cystolithotomy)

Indications

1. Stone larger than 2 cm.
2. Multiple stones.
3. Stone in a diverticulum (has thin wall, so easy to perforate).
4. Very hard stones.
5. Presence of another pathology needing surgery,
6. In children (weak vulnerable urethra).
7. Failure of crushing or disintegration.

Steps

1. Low midline incision or a Pfannenstiel's transverse incision.
2. The peritoneum is peeled upwards.

3. The bladder is identified by its trabeculated muscular wall and by the presence of a plexus of veins over its surface.
4. The bladder wall is held between two bladder forceps and it is opened.
5. The stone is extracted and the bladder is inspected for any associated pathology.
6. The bladder is closed in 2 layers with 2-0 polygalactin (Vicryl) after insertion of a suprapubic tube or a urethral catheter.
7. The prevesical space is drained and the wound is closed.

Treatment of urethral calculi

Stone in prostatic urethra

- A metal bougie is passed along urethra in an attempt to dislodge the calculus to the bladder where it is crushed by litholapaxy and the fragments are evacuated.
- If this procedure fails, the calculus is removed by open surgery through an incision in the bladder (suprapubic cystolithotomy).

Stone in membranous and bulbous urethra

- Removal of stone by urethral forceps or wire basket passed through a urethro-cystoscope.
- If the previous measures fail, open surgery is performed through a perineal incision. Wound is then closed on a urethral catheter.

Stone in penile urethra

Gentle trials to milk the stone out of the meatus. This may be helped by injecting a lubricant gel to the urethra. Gentle use of forceps may be done. No attempts is made to open on a stone in penile urethra for fear of penile urinary fistula because there is no muscle support of this part of the urethra.

Stone in fossa navicularis or at meatus

- Trial at milking out the stone.
- If milking fails, a meatotomy (cutting external meatus) is done to allow stone extraction.

Prevention of urinary stone recurrence

A person who formed a stone is liable to develop recurrent calculi.

General measures

1. Excess water intake: High fluid intake especially in hot weather. The patient should drink a large glass of water before going to bed and again if he wakes up in the middle of the night.
2. The stone should be chemically analyzed
3. Restriction of diet containing high chrystals according to chemical composition of stones.
4. Treating infections and other causes of stone formation.
5. Follow up of stone formers to detect early recurrences.
6. Metabolic work-up to know aetiology of stone.
    a. Serum calcium and phosphorus to exclude hyperparathyroidism.
    b. 24-hour urine collection for the following whose normal values are:
        i. Calcium <300 mg
        ii. Uric acid <800 mg
        iii. Oxalates < 40 mg
        iv. Citrates 300-900 mg

Calcium oxalate calculi

- Avoid diet rich in oxalates.
- Hydrochlorothiazide 50 mg bid aids in the dissolution of calcium oxalate crystals.
- Citrates 5 gm bid inhibits crystallization of oxalates.

Uric acid calculi

- Avoid diet rich in purines.
- Rule out myeloproliferative or neoplastic diseases.
- Urine should be kept alkaline, e.g., by sodium bicarbonate I gm t.d.s.
- Allopurinol (xyioric) 300 mg/day is indicated in patients with hyperuricaemia. It inhibits the formation of uric acid.

Ammonium magnesium phosphate (struvite)

- Aluminum hydroxide orally restricts phosphate absorption.
- Long term antibiotics to eradicate urinary tract infection.
- Avoid indwelling catheters.
- Increasing urine acidity by oral administration of 1g vitamin C daily.

Calcular anuria

This is a common serious and urgent problem in which there is arrest of urine flow (anuria) secondary to calcular obstruction of the ureter.

Aetiology

1. Stone obstructing the ureter of an only functioning kidney, the other kidney being non- functioning, surgically removed, or congenitally absent.
2. Bilateral renal or ureteric calculi.

Clinical features

1. Stage of onset. An attack of ureteric colic is followed by anuria, but sometimes, colic may be absent.
2. Stage of tolerance. Colic disappears and there no major sympyoms or signs. Still there is no urine output. Urea and creatinine start to rise.
3. Stage of uraemia (renal failure). Later, clinical picture of uraemia will supervene within a few days.

Differential diagnosis

Acute retention of urine. In contrast to retention, the urinary bladder is empty and not palpable in cases of anuria.

Investigations

1. Blood urea, creatinine and potassium are elevated to varying degrees according to the time of presentation.
2. A plain x-ray may be helpful as it may show a stone. Sometimes, it may be of no help as the stone is small, or radiolucent and the patient has gaseous distension.
3. Abdominal ultrasound is very useful. It reveals a distended pelvi-calyceal system on affected side. It should be remembered, however, that a large hydronephrotic kidney is usually not the side of acute obstruction as it is often nonfunctioning.

IVU is contraindicated as the patient is critically ill with high blood urea and creatinine.

Treatment (urological emergency)

1. Fixing a urethral catheter. A trial of forced diuresis during the investigations period is carried out by giving 40 mg frusimide in 100 ml Saline over 2 hours.

2. Drainage of the obstructed kidney should be done as early as possible. To clinically determine the side of obstruction, it is usually the side of recent ureteric or renal pain and the side with renal tenderness or guarding.
    a. Under anaesthesia a cystoscope is inserted and a ureteric catheter is passed up the affected ureter past the stone. If it succeeds a large amount of urine is drained. If the side of obstruction is not exactly known, both ureters should be catheterized.
    b. If ureteric catheterization fails, a percutaneous nephrostomy, performed under ultrasound or fluoroscopy guidance, will allow drainage of urine.
3. After renal function improvement the patient is investigated and diagnosed.
4. The stone is treated as mentioned earlier.
5. Following the relief of urinary obstruction, the patient may have severe diuresis where he may pass several litres of urine per day. This is due to the tubular defect caused by the obstructive process and due to the diuretic effect of high blood urea. This diuretic phase should be monitored and treated properly. The lost large amounts of fluid and electrolytes should be properly replaced.

Benign prostatic hyperplasia (BPH)

BPH is a normal aging process that affects most males. It is, therefore, also called senile prostatic enlargement.

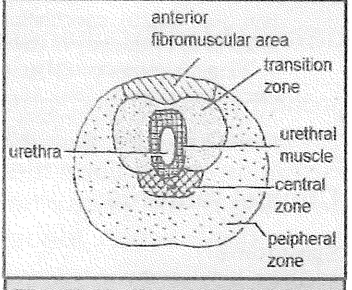


Fig. 46.18. Transverse cut section of the prostate showing its zones.

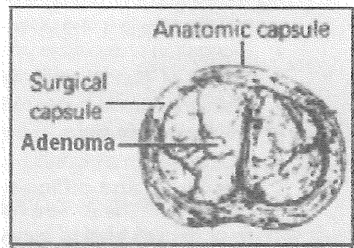


Fig. 46.19. Cross section BPH showing an adenoma compressing the surrounding prostatic tissue and forming a false capsule.

Incidence

- Enlargement of the prostate starts at age of 40 years.
- By the age of 50 years, about 50% of males have BPH.

Aetiology

The exact cause is not known but a sort of hormonal imbalance between androgens and estrogens, probably due to aging, stimulates transition-zone prostatic cells to undergo hyperplasia. Development of BPH is dependant on the presence of a functioning testis with adequate testosterone production.

Pathology

Microscopic picture

There is hyperplasia of glandular, fibrous and muscular elements with varying proportions.

Gross picture

Changes in the prostate

- The prostate is not divided into lobes but is actually divided into zones that are morphologically and functionally different (Fig. 46.18).
    - Anterior fibromuscular stroma.
    - Peripheral zone.
    - Central zone.
    - Transition zone (periurethral area). It is the transition zone which is the origin of BPH (on contrary to prostate cancer that originates from the peripheral zone).
- BPH starts by development of nodules that enlarge, compress urethra and rest of the prostate. These nodules, later, form the main bulk of BPH tissue and the remaining

compressed prostatic tissue forms prostatic surgical capsule within which the adenoma is enucleated during surgery (Fig. 46.19).

- BPH is formed of nodular tissue that is termed "adenoma".
- If hyperplasia is more prominent on both sides of the urethra, BPH is described as enlargement of lateral lobes. If, on other hand, the hyperplastic tissue is central and grows up elevating the trigone, it is described as middle lobe enlargement (Fig. 46.20). In some cases enlargement may be trilobar.

Urethral changes

The adenoma compresses the urethra above the verumontanum producing:

1. First, urethral elongation.
2. Urethral narrowing as it is stretched and compressed from side to side. It becomes an antero-posterior slit. This narrowing interferes with bladder emptying.
3. Exaggeration of the normal posterior curve of urethra, so that it interferes with a passage of catheter or an instrument. A special (coudé) curved catheter or instrument can pass to bladder, if needed.

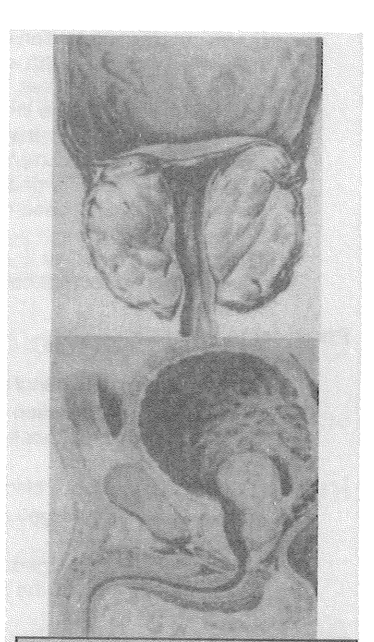


Fig. 46.20. Enlargement of lateral lobes (upper) and of middle lobe (lower). Notice the bladder trabeculations.

Urinary bladder changes (Fig. 46.1)

The urinary bladder suffers from chronic obstructive uropathy mentioned before in the form of hypertrophy, later, trabeculation (Fig. 46.20), sacculation and diverticula formation. In addition there is detrusor hyper-reflexia leading to frequency of micturition. The bladder does nor empty completely, which leads to post- voiding residual urine (Fig. 42.10).

Ureters and kidneys (Fig. 46.1)

At a later stage, the ureters and kidneys may suffer from the back-pressure changes that were mentioned earlier, e.g., bilateral hydroureter and hydronephrosis.

Clinical features

Symptoms

There is no correlation between the size of the prostate and the degree of symptoms. Symptom severity actually depends upon the degree of urethral and bladder neck obstruction by the enlarged gland. BPH may be asymptomatic.

1. **Frequency and urgency of micturition** are the usual early complaints, At first, frequency is mainly nocturnal and the patient has to wake up two or three times by night to void. Later, frequency becomes progressive and occurs night and day. Frequency is due to:
   a. Elongation of sensitive posterior urethra exposes its mucosa to urine in the bladder.
   b. Stretch of internal sphincter allows a few drops of urine to escape in the posterior urethra.
   c. Detrusor muscle hyper-reflexia increases frequency.
   d. Residual urine starts to accumulate in bladder.
   e. Complication as cystitis, diverticulum or stone of urinary bladder.
2. **Hesitancy and difficulty in micturition**. There is difficulty to start, to maintain, and to terminate micturition. The patient has to wait for a few seconds to start

micturition (hesitancy). Any attempt to strain will lead to congestion of the prostate with more obstruction, hence the patient has to relax in order to void.

3. **Weak and diminished calibre of urine stream.** Urine stream is not forceful. There is inability to terminate micturition abruptly as the patient has post-voiding dribbling.
4. **Acute retention of urine** is sometimes the first presentation of BPH. Retention is precipitated by excess fluid in take, diuresis, constipation, cold weather, alcoholic drinks, cystitis, prolonged confinement to bed, unreleived sexual excitement and drugs (e.g., diuretics, antispasmodics or antitussives). Acute retention is very painful and needs urgent intervention.
5. **Chronic retention with overflow incontinence**. As prostatic obstruction progresses, more residual urine accumulates in the bladder until a time comes when it becomes overdistended and may contain up to one litre of urine. The bladder turns atonic and the patient involuntarily voids small amounts when intravesical pressure exceeds intraurethral pressure. The condition is painless and actual complaint of the patient is incontinence.
6. **Haematuria**. An enlarged prostate may compress the prostatic venous plexus resulting in congestion of veins at base of the bladder. Gross haematuria may occur due to rupture of one of these veins.
7. **Sexual symptoms**. At first, the patient may have increased libido. Later, impotence may occur.
8. **Symptoms of complications**, e.g., bladder stone, cystitis, bladder diverticula or chronic renal failure.



Fig. 46.21. IVUs showing smooth elevation of bladder base that is caused by BPH. The lower shows the fish-hook sign in the right ureter.

Physical examination

General examination

- Evidence of chronic renal insufficiency.
- Neurological examination is needed to exclude a neurogenic bladder (commonest cause is diabetic autonomic neuropathy).

Abdominal examination

- Suprapubic mass (full bladder) may be felt.
- Kidneys may be enlarged and palpable.

Digital rectal examination

The prostate is felt enlarged, smooth and firm. The median sulcus is preserved and the rectal mucosa can be moved over the prostate.

Investigations

1. Routine laboratory tests. Urine analysis, CBC, blood urea, creatinine and electrolytes.
2. **Serum PSA** (prostatic specific antigen) to exclude associated prostate cancer. Normal value is 0-4 ng/ml.
3. **Flowmetry**. Maximum flow-rate less than 15 ml/sec. is indicative of obstruction.
4. **KUB**. May show bladder or prostatic stones.
5. **Abdominal sonography** to visualize kidney changes, measure amount of post-voiding residual urine in urinary bladder, and to diagnose any bladder pathology.

6. **IVU**. The following changes may be evident with BPH:
   a. Smooth basal filling defect in the bladder (Fig. 46.21).
   b. Trabeculations, sacculations and diverticula.
   c. Hooking of ureters on entry into bladder (fish-hook sign, Fig. 46.21).
   d. Large amount of postvoiding residual urine.
   e. Hydronephrotlc changes of the kidneys (Fig. 46.3).
7. **TRUS** (trans-rectal-ultrasound)
   a. To uncover non-palpable malignancy in peripheral zone. Ultrasound guided biopsy can be taken from the suspicious area.
   b. To assess size of enlargement and its pattern, i.e., middle lobe, bilobar, or trilobar.
8. **Cystourethroscopy** is indicated in patients presenting with haematuria to exclude bladder pathology.

- BPH is a normal aging process and not a disease by itself unless an obstruction starts to develop. Its treatment when complicated is by TURP in most of the cases.
- BPH and prostate cancer can be present in the same patient.
- PSA measurement is mandatory in all men above the age of 50 for early detection of prostate cancer.

Treatment

Medical treatment and watchful waiting

Since natural history of BPH is not uniform, patients with minimal symptoms and no complications may remain as such for years. These patients may be managed by:

- **Watchful waiting**. The patient is advised to avoid diuresis, constipation, wine, unrelieved sexual excitement and withholding micturition for long periods. Close follow-up with periodic flowmetry and symptoms score.
- **Medical treatment**
  - Prostatic decongestant suppositories.
  - Alpha-blockers, e.g., trasocine or prasocine. These block a- receptors in bladder neck and prostatic urethra so diminish outlet muscle tone. They improve symptoms, particularly frequency, and flow-rates.
  - 5-alpha-reductase inhibitors, e.g., proscar. This drug inhibits the enzyme 5-alpha-reductase that changes testosterone to active dihydrotestosterone in the prostate. In those who improve on this treatment, the drug is continued for life.

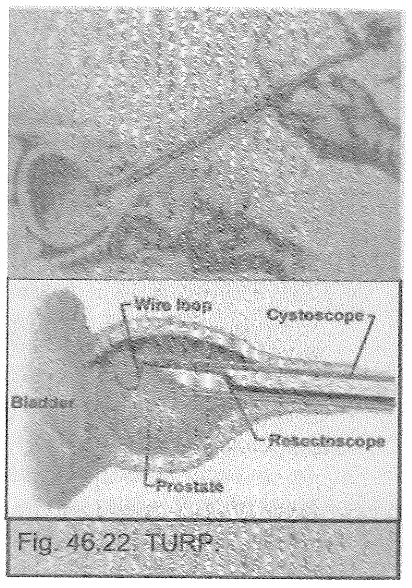
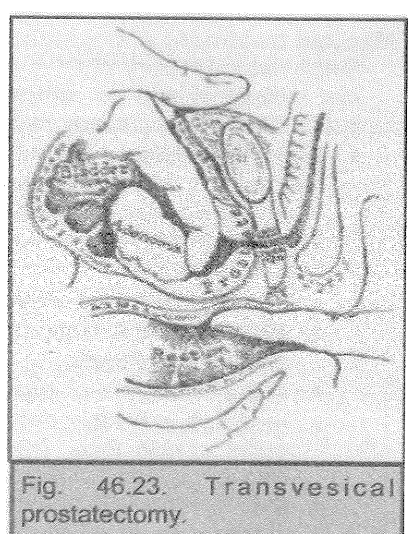


Fig. 46.22. TURP.


Fig. 46.23. Transvesical prostatectomy.

Retention of urine. Refer to chapter 42.

Minimally invasive procedures

The aim of these techniques are temporary treatment of BPH (tunneling) when definitive treatment is contraindicated in high risk patients. These include:

- Thermotherapy. Microwave heat therapy is applied by special probes per urethra or per rectum.

- Endoscopic transurethral cryo-ablation of the prostate.
- Endoscopic transurethral prostatic stents.

Definitive surgical treatment

The idea of surgery is to remove adenomatous gland enlargement from inside its false capsule of compressed prostatic tissue, which is left unremoved.

Indications for surgery

1. Obstructed uroflow curves.
2. Acute retention of urine.
3. Chronic retention with residual urine more than 200 ml.
4. Haematuria.
5. Complication as bladder diverticula or stones.
6. Back pressure effects on upper urinary tract.
7. Distressing frequency of micturition.

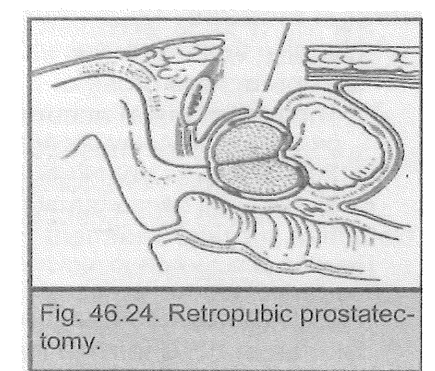


Fig. 46.24. Retropubic prostatectomy.

Techniques of surgery

- **Endoscopic transurethral surgery**
    - Trans-urethral resection of the prostate (TURP) is the standard technique. Using a cysto-resectoscope the adenoma is removed trans-urethrally piece by piece, using an electrocautery (Fig. 46.22). The only limitation is a large adenoma making the procedure lengthy and hazardous.
    - Visual laser ablation of the prostate (VLAP).
    - Transurethral heat prostatic vaporization.
- **Open surgical techniques** are needed for exceptionally large adenomas.
    - Transvesical prostatectomy. This operation is indicated when there is an associated bladder pathology that requires surgery, e.g., diverticulum or big stone. Through a midline or Pfannenstiel incision the bladder is opened and the adenoma is enucleated from within its lumen (Fig. 46.23). After haemostasis the bladder is closed over a suprapubic cystostomy tube.
    - Retropubic (Millin's) prostatectomy. Through a similar incision, the retropubic space is exposed, but the bladder is not opened. A transverse incision is done directly through capsule of the prostate (Fig. 46.24). The adenoma is enucleated and haemostasis is secured under direct vision by suture and diathermy. Foley catheter is inserted and prostatic capsule is closed with no suprapubic tube.

Complications of surgery

1. Bleeding is the main problem. It may be primary, reactionary or secondary. Clot retention may occur.
2. Retrograde ejaculation due to damage of the internal sphincter. It occurs in more than 90% of cases.
3. Infection leading to urethritis, cystitis or epididymo-orchitis.
4. Bladder neck fibrosis.
5. Urethral stricture.
6. Urinary incontinence. True incontinence from sphincteric injury is very rare. It occurs in 1/10,000 operation. Temporary urge and stress incontinence for a few days are, however, common.
7. TURP syndrome. It occurs only with endoscopic resection of the prostate and is due to over absorption of irrigating hypotonic fluid used during endoscopy It is characterized by hypervolaemia, dilutional hyponatraemia and intravascular

haemolysis. The use of glycine (isotonic solution) for irrigation avoids this dangerous complication.

Bladder diverticula

A diverticulum is a flask shaped pouch of bladder wall. It may be congenital, but most are acquired due to distal obstruction to bladder or urethra (infravesical obstruction).

Clinical features

- Usually there are no specific symptoms to indicate the presence of a diverticulum. Symptoms are those of the obstructive lesions.
- Double micturition occurs with large diverticula. The diverticulum fills with urine during micturition and subsequently empties into the bladder. This causes an immediate desire to pass urine for a second time.
- Complicated diverticula give rise to frequency, pain, and haematuria.

Complications

- Rarely it may obstruct the ureter.
- Stones may be formed inside.

Diagnosis

- Diagnosis of diverticula should be accompanied by diagnosis of their cause.
- IVU, ascending cystography (Fig. 46.25), and cystoscopy are all helpful.

Treatment

- Treatment of the cause is mandatory.
- Diverticula are not excised unless complicated. The operation is usually done in the course of surgery for distal obstructive lesion, e.g., with prostatectomy.

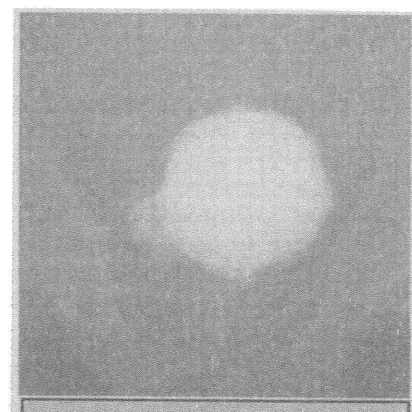


Fig. 46.25. Ascending cystogram showing multiple bladder diverticula.

Urethral stricture

Urethral stricture usually affects young active males. It is very rare in females.

Aetiology

Most common causes are post-traumatic strictures and gonococcal urethritis (Fig. 46.26).

1. Congenital causes, e.g., meatal stenosis. True congenital urethral stricture is very rare.
2. Inflammatory
   a. Post-gonococcal is the commonest inflammatory cause. It affects the bulbar urethra in 70% of cases.
   b. Prolonged urethral catheterization.
3. Post-traumatic (chapter 44) is by far the most common cause.
4. Neoplastic.

- A posterior urethral valve is not a true stricture as a catheter passes easily up to the bladder.
- Urethral stricture is mainly traumatic and less commonly gonococcal. Its treatment is mainly dilatation with or without surgery.

Pathology

Types

1. Passable strictures allow passage of urine and instruments.
2. Permeable strictures allow passage of urine but no instruments can pass.

3. Impermeable strictures where neither urine nor instruments can pass.

Complications

1. Prostatitis and epididymo-orchitis.
2. Secondary infection with periurethral abscess formation.
3. Urethral urinary fistula following spontaneous rupture or drainage of a periurethral abscess.
4. Infertility (from the trauma, surgery or obstructed semen flow.
5. Complications of obstructive uropathy, i.e., back pressure changes, infection, stone formation, and renal failure.

Clinical features

1. Difficult micturition. The patient has to strain in order to pass urine (in contrast to BPH where the patient has to relax). The stream is thin and weak. Terminal dribbling may occur as urine trickles from the dilated proximal urethra.
2. Urethral discharge may be present because of infection.
3. Frequency due to incomplete bladder evacuation or due to cystitis.
4. Acute retention is precipitated by excess water or by activation of local infection.
5. Chronic retention occurs in long-standing cases and causes bladder fullness and possibly dribbling due to retention with overflow.
6. Manifestations of obstructive uropathy. In advanced cases all changes mentioned earlier may occur.

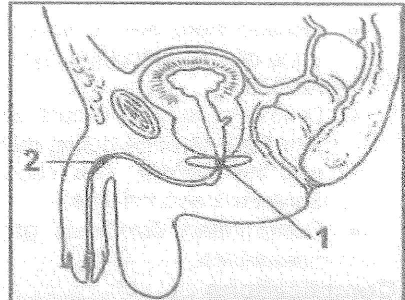


Fig. 46.26. Common sites and causes of urethral stricture:
1. Post-traumatic.
2. Post-gonococcal.

Investigations

1. Uroflowmetry reveals obstructed flow characterized by straining.
2. Ascending urethro-cystography (Fig. 46.27) detects the site, length, degree and shape of stricture.
3. Urethro-cystoscopy to see the stricture and assess its possible treatment.
4. Sonography to assess the upper urinary tract.
5. IVU to assess renal function and back pressure changes.



Fig. 46.27. Urethro-cystography showing a stricture of bulbar urethra.

Treatment

- **Urethral dilatation**. Under absolute sterile conditions and after instillation of a local anesthetic in the urethra, dilatation is performed very gently. Dilatation usually starts with gum elastic bougies, or even fihiform bougies (Fig. 46.28) for very narrow strictures. Metal bougies can be used later.
- **Endoscopic visual internal urothrotomy**. Through a urethroscope and under direct vision, the stricture is visualized and incised with a sharp knife blade. Internal urethrotomy is followed by regular dilatation.

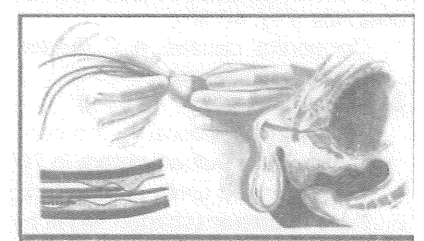


Fig. 46.28. Dilatation of a very tight urethral stricture using filiform bougies.

- **Surgical urethroplasty** is done for strictures of bulbar and penile urethra if previous measures fail. The idea is to excise the fibrous tissue of a stricture and to do end to end anastomosis. If the stricture is long a tube or a flap is constructed from penile skin, scrotum, or perineum and is used to replace the strictured segment.

TUMOURS OF THE URINARY TRACT

CHAPTER CONTENTS

Renal neoplasms

For practical reasons most of the neoplasms of the kidney are malignant. Tumours of the kidney can be divided in two main groups.

1. Tumours arising from the renal parenchyma (90%).
   a. Renal cell carcinoma
   b. Nephroblastoma
2. Tumours arising from the renal pelvis (10%).
   a. Transitional cell carcinoma.
   b. Squamous cell carcinoma

Renal cell carcinoma (hypernephroma)

This is the commonest renal parenchymal tumour. It accounts for 75% of all renal neoplasms.

Incidence

- Male to female ratio is 2:1.
- The majority of the patients are 50-70 years of age.
- The tumour is usually unilateral. Bilateral tumours are either synchronous or metachronous and occur in 1-2% of cases only.

Aetiology

1. There is a two fold increased risk in persons who use tobacco products.
2. A higher incidence is noted in patients with Von-Hippel Lindau disease in which there may be renal and pancreatic cysts, cerebellar haemangioblastomas, retinal angiomata and pheochromocytoma. Renal cell carcinoma in these patients may be multiple and bilateral.
3. Loss of the short arm of chromosome 3 is a consistent chromosomal change.

Pathology

Gross appearance

- The tumour usually starts in one pole of the kidney.
- It ranges in size from a few centimeters to lesions which fill the abdomen.
- The colour of the tumour is golden yellow due to its high lipid content.
- Areas of haemorrhage and necrosis are common giving the tumour a mosaic appearance.
- There is an apparent false capsule surrounding the lesion but the tumour actually invades the adjacent tissues beyond it.
- The tumour infiltrates the renal pelvis early in the course of the disease.

Microscopic appearance

- The tumour is an adenocarcinoma that arises from the cells of the proximal convoluted tubules.

- The majority of the lesions exhibit clear cell pattern as the cholesterol crystals in the cytoplasm are dissolved during preparation. A granular cell type is due to the increased mitochondria in the cytoplasm.

Spread

Direct

- The neoplasm soon infiltrates the renal pelvis and this explains the early and common occurrence of haematuria,
- It remains localized to the renal capsule for sometime but eventually it infiltrates it then the Zukercandle's (Gerota's) fascia and may even infiltrate adjacent organs.
- The tumour may invade and grow inside the renal vein, then the inferior vena cava (IVC).

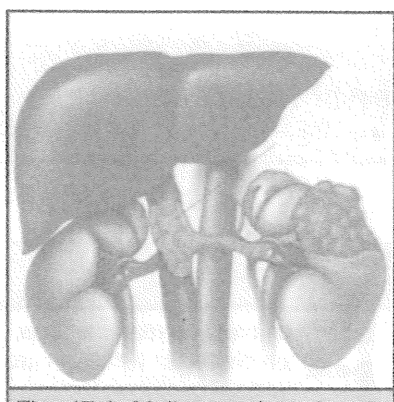


Fig. 47.1. Malignant thrombus in left renal vein and IVC.

Lymphatic

Spread occurs to lymph nodes along the hilum of the kidney, then to the para-aortic nodes.

Blood

Haematogenous spread gives rise to metastases in the lungs, bones, and brain. Sometimes, a malignant thrombus may be present in the renal vein or extends even to the inferior vena cava (Fig. 47.1).

Clinical features

1. **Haematuria** is present in 50% of cases. It is classically described as being painless, recurrent, profuse and total haematuria. Sometimes, the blood passes as tubular clots taking the shape of the ureter.
2. **Pain** is present in 40% of patients. It may be due to:
   a. Stretch of the renal capsule by the neoplasm.
   b. Passage of blood clots causing Ureteric colic.
   c. Infiltration of adjacent lumbar nerves causing referred pain.
3. **Renal mass**. An irregular hard, renal swelling can be felt in 30% of cases. The classical triad of haematunia, pain and renal mass is present in 10% of cases only. The triad usually indicates an advanced disease.
4. **Metastases**. Sometimes, the first presentation of the patient is the presence of metastases, e.g., pulmonary or skull deposits, and only after investigations, the presence of renal cell carcinoma is detected.
5. **Non-specific symptoms as fever**, night sweats, weight loss or anaemia.
6. **Secondary varicocele**. A rapidly enlarging varicocele which dose not empty by elevation of the scrotum may suggest the presence of a renal carcinoma.
7. **Systemic syndromes**
   a. Hypercalcaemia occurs in 5% of cases due to secretion of a parathormone-like substance by the tumour or due to the presence of bone metastases.
   b. Polycythaemia.
   c. Amyloidosis.

Investigations

1. Plain X-ray (KUB) may show mottled central calcification.
2. IVU may reveal:
    a. Enlargement of the kidney.
    b. Elongation, displacement, compression or amputation of a calyx (Fig. 47.2).
    c. Displacement of the renal pelvis.
    d. Rarely, a non functioning kidney.
3. Renal sonography shows the tumour as a solid mass.
4. CT scan is mandatory in suspected cases. It is very accurate in detecting:
    a. The exact site, size and consistency of the lesion.
    b. Invasion of the renal capsule or any surrounding tissues.
    c. Lymph node enlargement and invasion of the renal vein or inferior vena cava.
5. Angiography was in the past frequently used to diagnose hypernephromas by visualizing malignant circulation. It has now been replaced by CT scan.
6. Chest X-ray and isotope bone scan for metastases.
7. Staging (Table 47.1)



Fig. 47.2. IVU showing amputation of the left upper calyx in a case of renal cell carcinoma.



Fig. 47.3. CT scan showing left renal cell carcinoma.

Treatment

For early operable cases (stages I & II) surgery offers the only hope for cure. Radical nephrectomy is done. It entails removal of the kidney within its sheath of Gerota's fascia together with the ipsilateral adrenal gland. It is recommended to expose and ligate the vascular pedicle as a first step of the operation for:

- Prevention of dissemination of malignant cells during manipulation of the tumour.
- To have good control on a very vascular tumour.
- To be able to remove a malignant thrombus from the inferior vena cava, if present.

Table 47.1. Stages of renal cell carcinoma.

Stage	Description
I	Tumour confined to the kidney
II	Tumour confined to Gerota's fascia & involves the perinephric fat
III	Tumour involves the renal vein and/or regional lymph nodes
IV	Distant metastases

Advanced disease

Patients who have metastatic disease may receive symptomatic treatment, e.g., analgesics, irradiation and immunotherapy using alpha or gamma interferons and interleukin-2.

Wilms' tumour (nephroblastoma)

This tumour arises from embryonic nephrogenic tissue and contains both epithelial and connective tissue elements.

Incidence

- Nephroblastomas account for 10%. Of childhood malignant tumours and 80% of all genitourinary cancers in children under 15 years of age.

- Peak incidence is 3 to 4 years of age. 90% of cases occur before the age of 7 years.
- Male to female ratio is 1:1.

Pathology

Gross appearance

- The tumour appears as a solitary sharply demarcated, encapsulated mass.
- Haemorrhage or necrotic areas are present.
- Renal pelvis invasion is rare.
- Bilateral tumours are present in 5-10% of cases.

Microscopic picture

1. The tumour contains both epithelial and connective tissue elements. Epithelial cells may form primitive glomeruli or tubules. Connective tissue elements as cartilage, fat, smooth or striated muscles are present.
2. The degree of tumour differentiation varies from favourable histology (FH) to unfavourable histology (UH) where there are marked variations in nuclear size with hyperchromatism and excessive mitoses.

Anomalies that *may* be associated with Wilms' tumour

1. Aniridia in 2% of cases.
2. Hemihypertrophy. Asymmetrical growth of one side of the body in 3% of cases.
3. Macroglossia
4. Thirty fold increase in the incidence of neurofibromatosis.
5. Genito-urinary anomalies in 4.4% of cases as renal hypoplasia, hypospadias or cryptorchidism.

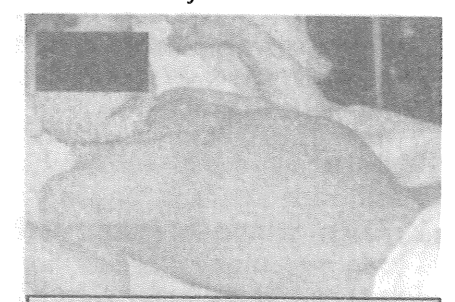


Fig. 47.4. Wilms' tumour usually presents as a large abdominal mass in a child.

Clinical features

1. The main presentation (90%) is an abdominal mass (Fig. 47.4), which is smooth, firm and is confined to one side of the abdomen.
2. One third of patients present with vague abdominal pain, often associated with minor trauma and haemorrhage within the tumour.
3. Microscopic haematuria occurs in 50% of cases.
4. Hypertension is present in up to 60% of cases. It results from encroachment on the blood supply producing renal ischaemia and excess renin production.

Nephroureterectomy has two indications

1. **TCC of renal pelvis.**
2. **Renal tuberculosis if the kidney is damaged beyond cure by anti-tuberculous therapy.**

Differential diagnosis

1. Neuroblastoma is also a childhood tumour that greatly simulates nephroblastoma. It usually forms an irregular tumour which may cross the midline.
2. Renal swellings
   a. Hydronephrosis.
   b. Multicystic dysplastic kidney.
   c. Infantile type of polycystic kidney.

Staging

1. Stage I: Tumour limited to the kidney and completely excised.

2. Stage II: Tumour beyond the kidney and completely excised.
3. Stage III: Residual tumour
4. Stage IV: Metastases
5. Stage V: Bilateral tumours

Investigations

Laboratory

1. Complete blood count, liver and renal function tests.
2. Urine catecholamines are within normal. They are elevated in case of neuroblastoma.

Imaging

1. IVU reveals an enlarged kidney, the pelvicalyceal system is greatly attenuated and distorted. The kidney may be non-functioning.
2. Ultrasonography should be routinely done. It can distinguish solid renal masses from hydronephrosis and renal cystic disease. It can also detect small tumours and liver metastases.
3. CT scanning can also differentiate cystic from solid masses. It can detect involvement of adjacent structures or involvement of the other kidney. Furthermore, CT can follow the tumour response to chemo and radiotherapy.
4. Chest x-ray and isotope bone scan to detect metastases.

Treatment

Surgery

Surgical excision remains the cornerstone of therapy in all children with Wilms' tumour.

- For operable tumours the affected kidney is radically resected as for renal cell carcinoma.
- For large unresectable lesions
    - A course of preoperative chemotherapy usually shrinks the tumour, which can then be removed.
    - Remaining tumour in nodes or adjacent tissues should be marked with surgical clips to facilitate direction of radiation therapy.

Post-operative treatment

- If there is no residual tumour, adjuvant chemotherapy using actinomycin D, vincristin and adriamycin, is given.
- If there is residual tumour, radiotherapy is added for local control.

Prognosis

Chemo and radiotherapy have improved the overall prognosis to 80% 5-year survival. Early cases are usually cured.

Tumours of the renal pelvis

Transitional cell carcinoma (TCC) accounts for 7% of renal tumours

Predisposing factors

1. Tobacco use leads to 4 folds increase in the risk of developing TCC of the upper urinary tract.
2. Phenacetin abuse. Long-term, high-dosage intake of the drug increases the risk.

Pathology

1. TCCs are collectively called urothelial tumours as they arise from the transitional epithelium that lines the renal pelvis, ureter, and the urinary bladder.
2. TCCs vary in malignancy from low-grade malignancy (which was formerly called papilloma) to highly anaplastic tumours.
3. A patient with a TCC of the renal pelvis is likely to develop the same lesion in the ureter, the bladder or even the contralateral renal pelvis. This is explained by either:
    - Multicentricity of this neoplasm, where the whole urothelium is predisposed to malignancy by a certain carcinogen.
    - Trans-luminal spread of shedded malignant cells.

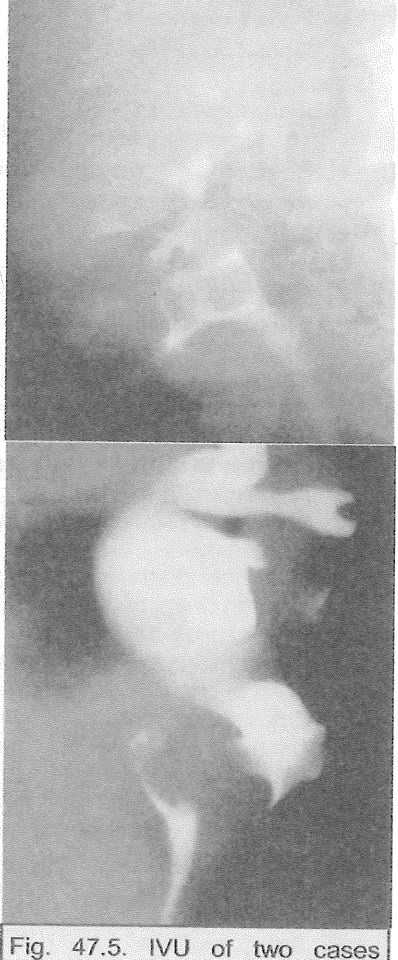


Fig. 47.5. IVU of two cases showing irregular filling defects of the renal pelves in cases of TCC.

Clinical features

1. Gross or microscopic haematuria is present in 90% of cases.
2. Obstruction causes renal pain and/or renal swelling (hydronephrosis).

Investigations

1. Intravenous urography (IVU). The radiograph shows a fixed irregular radiolucent defect (Fig. 47.5) or non-visualization of part or all of the collecting system.
2. CT scan helps to differentiate renal parenchymal from renal pelvic tumors. It can visualize lymph node deposits.
3. Cytology (microscopic examination of urine for malignant cells)
4. Flexible ureteropyeloscopy. A flexible endoscope is introduced from the bladder to inspect the whole upper tract. Any lesion is visualized and biopsied.

Treatment

The classic therapy is nephroureterectomy with excision of an adjacent cuff from the bladder. If a ureteral stump is preserved, there is a 50% chance for stump recurrence, which is difficult to monitor.

Squamous cell carcinoma

- This tumour occurs on top of leukoplakia. It is often associated with infection and calculus disease.
- It forms a nodular ulcerated mass, which invades the renal pelvis and parenchyma, and spreads to the neighbouring structures, but surface implantation does not occur.
- Clinically, there is haematuria. In late cases, extrarenal spread causes severe pain.
- The tumour has a bad prognosis.

Carcinoma of the urinary bladder

- This is the commonest urologic malignancy in Egypt.

- In the past, discussion of bladder carcinoma used to be based upon whether it was of Bilharzial or nonBilharzial origin. Nowadays, the histologic type of the tumour (squamous cell carcinoma or transitional cell carcinoma) is the principal differentiation.
- The "Registry of the Department of Urology at Cairo University Hospitals" shows that relative incidences of bladder cancer types in Egypt are:
    - 55% transitional cell carcinoma.
    - 40% squamous cell carcinoma.
    - 5% others including adenocarcinoma.

Squamous cell carcinoma (SCC)

Aetiology

1. This type usually occurs on top of long-standing Bilharzial cystitis that is complicated by secondary bacterial infection. Many theories are suggested to explain the possible role of Bilharzial cystitis as a predisposing factor:
    a. Mechanical irritation. Continuous irritation of the bladder mucosa is caused by the ova as they pass through the bladder wall to reach the urine.
    b. Chronic infection especially by ammonia producing organisms may lead to squamous metaplasia.
    c. Increased level of B-glucuronidase enzyme in patients with Bilharzial cystitis especially in the presence of bacterial infection. This enzyme transforms non carcinogenic compounds as hydroxy B-naphthylamine glucuronide to a carcinogenic compound.
2. Abnormalities of tryptophan metabolism that lead to the production of carcinogenic compounds,
3. N. nitroso compounds. Nitrates are present in many vegetables and in drinking water if it is contaminated by nitrate fertilizers. These nitrates are excreted in urine where they are acted upon by bacteria leading to the liberation of N. nitroso compounds which are carcinogenic,

Pathology

Preneoplastic alterations of the urothelium

1. Brunn's nest are clusters of benign-appearing transitional cells situated in the submucosa.
2. Cystitis cystica is Brunn's nest that have undergone central liquefaction.
3. Cystitis glandularis is similar to cystitis cystica except that the transitional cell lining of the cysts has undergone glandular metaplasia, i.e., changed to columnar epithelium.
4. Squamous metaplasia is a proliferative lesion in which the normal transitional epithelium is replaced by a mature, non keratinizing squamous epithelium.
5. Leukoplakia is the presence of mucosal well-defined, thick and raised white patches. Microscopically there is squamous metaplasia and marked keratinization with cellular atypia and dysplasia.

Gross appearance

- Nodular fungating type (commonest).
- Malignant ulcer.
- Papillary tumor (rare).

The tumour usually arises from the posterior and lateral walls of the bladder.

Microscopic appearance

1. The tumour is composed of keratinized cells and usually contains concentric aggregates of cells that are called squamous pearls (cell nests).

2. Squamous cell cancers shed cells in urine that can be detected cytologically in most patients.

Spread

1. Direct. The tumour infiltrates the wall of the bladder and then may infiltrate adjacent organs as the seminal vesicles and prostate in males or the vagina in females.
2. Lymphatic. The incidence of lymph node metastases is 15%.
3. Blood borne spread is rare.



Fig. 47.6. Descending cystogram (part of IVU) showing an irregular filling defect in the urinary bladder that is suggestive of bladder cancer. Cystoscopy and biopsy are needed for confirmation.

Clinical features

The male to female ratio is 4:1 and the peak age incidence is between the third and fifth decades.

Symptoms

1. The patient usually presents by recent exaggeration of the symptoms of cystitis which have been present for a long time. There is burning micturition, frequency and pyuria.
2. There may be recurrent attacks of painful haematuria.
3. Necroturia means the passage of pieces of whitish tissue representing necrotic tumour.
4. There may be suprapubic pain.
5. Rarely, the tumour may cause urine retention.

Signs

1. Generally the patient may have pallor (anaemia) and signs of renal failure.
2. Digital rectal examination (DRE) and bimanual examination will assess the size, site and mobility of the tumour.

Wallace staging for bladder cancer is based on bimanual clinical examination.

T_0 No palpable mass.

T_1 Palpable mobile mass, but no induration of the bladder wall.

T_2 Palpable mass with induration of the bladder wall.

T_3 Palpable mobile wall with extravesical spread.

T_4 Fixed bladder mass.

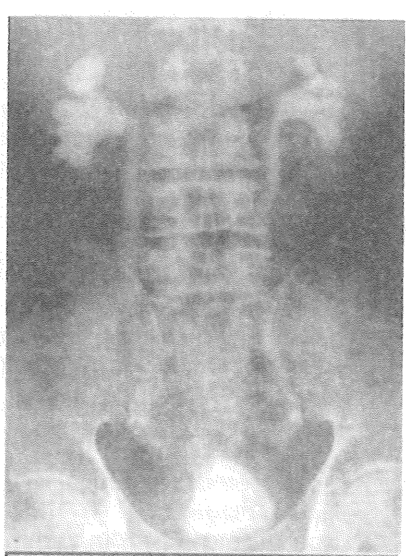


Fig. 47.6. IVU showing an irregular filling defect in the bladder with bilateral hydroureter and hydronephrosis. The kidneys, however still function fairly well as shown by excretion and concentration the contrast material.

Investigations

Laboratory tests

- Urine examination for RBCs, and pus cells.
- Urine cytology for malignant cells.
- Blood urea and creatinine and liver function tests.

Imaging

- IVU will reveal the presence of the lesion and will assess renal function (Fig. 47.6 & 47.7).

- Abdominal and pelvic ultrasound can assess the kidney size and the bladder mass.
- CT scan can assess the extent of the tumour and any pelvic node deposits.
- Cystoscopy and biopsy are mandatory.

Treatment

Unfortunately in Egypt by the time the diagnosis of squamous cell carcinoma of the urinary bladder is made, the case is too advanced. Attempts to cure these patients by transurethral resection (TUR) or by partial cystectomy are not feasible. SCC is also resistant to chemo and radiotherapy, and so the best chance for the patient is to do a radical cystectomy operation.

In males this procedure entails resection of the whole bladder with its perivesical fat, the prostate, the seminal vesicles and the lower ends of both ureters. In the female the urinary bladder, the uterus, the tubes and the anterior vaginal wall are removed. Block dissection of the internal and external iliac and obturator lymph nodes is perfomied in both sexes. Urinary diversion is then performed.

Prognosis

The 5-year survival following radical cystectomy is 32%.

Transitional cell carcinoma (TCC)

Aetiology

1. Industrial carcinogens
    a. Aniline dyes, petrol and leather industries.
    b. Rubber and textile industries due to exposure to aromatic amines.
2. Cigarette smoking results in four-fold increase in risk.
3. Cyclophosphamide (cancer chemotherapeutic drug) results in nine-fold increase in risk.
4. Pelvic irradiation. Four-fold increase in risk for developing bladder cancer is noted in women treated for cervical malignancy.
5. Prolonged intake of large doses of phenacetin.

Pathology

Gross appearance

- Any part of the bladder may be involved but the common sites are the base, trigone and around the ureteric orifices.
- They are often multiple.
- Gross types
    - Localized erythematous patches.
    - Papillary lesions (Fig. 47.8).
    - Nodular lesions.
    - Malignant ulcers.

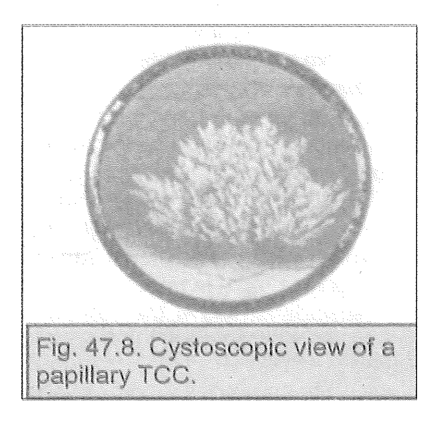


Fig. 47.8. Cystoscopic view of a papillary TCC.

Microscopic picture

- According to the degree of cellular differentiation, TCC is classified into:

Grade I	Well differentiated
Grade II	Moderately differentiated
Grade III	Poorly differentiated

- According to the depth of invasion of the bladder wall, TCC is classified into:
    - Superficial TCC. These lesions are usually papillary. There is no invasion of the muscle layer. 25% of these lesions will later display muscle invasion or subsequent recurrent lesions.

- Muscle invasion TCC. These are usually solid. There is invasion of the muscle layer.

Investigations

- As in SCC.
- In addition transurethral resection of visible tumour with biopsy of the underlying bladder muscles is done to do proper staging. Random biopsies are also taken to diagnose carcinoma in situ.

Treatment

Superficial bladder cancer

1. Transurethral resection (TUR), excising the tumour with the underlying muscle plus multiple random biopsies to detect carcinoma in situ. Laser ablation may be used in place of TUR.
2. Intravesical chemotherapy (adriamycin) or immunotherapy (BCG vaccine) is used after TUR to prevent recurrence in grade II and III tumours and in the presence of carcinoma in situ.
3. Regular follow up for 5 years is indicated to detect recurrence. If there are multiple recurrences or persistent carcinoma in situ, the patient is treated by radical cystectomy.

Muscle invasive TCC

1. Radical cystectomy. This operation differs from that described for SCC in only one step; the urethra has to be removed because of the tendency of TCC to spread along the urethra.
2. Radiotherapy. The results are inferior to surgery.
3. Chemotherapy for metastatic disease or when the surgical margins are not free. Best combination is methotrexate, vinblastine, adriamycin and cis-platin "MVAC".

Urinary diversion following cystectomy

Many methods are available; the choice depends upon the condition of the patient and the surgical experience.

Ureterocolic anastomosis (Fig. 47.9)

The two ureters are anastomosed to the sigmoid colon. The operation is simple, rapid and the patient is continent.

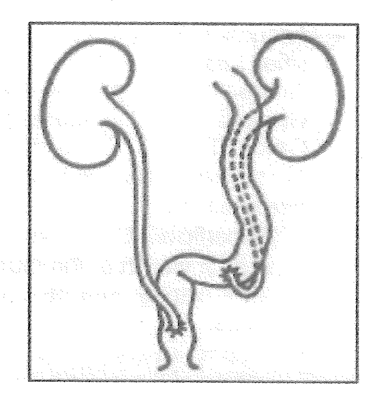


Fig. (47.9): Ureterocolic anastomosis

Disadvantages

1. Recurrent upper urinary tract infections.
2. Hyperchloraemic acidosis; as chlorides in urine can be absorbed by the colonic mucosa. Another factor is loss of bicarbonate ions as urine irritates the intestinal mucosa leading to outpouring of the alkaline intestinal secretions, To minimize this complication, the intake of sodium chloride is restricted and constipation is avoided.
3. Hypokalaemia as intestinal secretions are rich in potassium.
4. Deterioration of renal functions.
5. Encephalopathy. Urea in urine is acted upon by the intestinal flora leading to the formation of ammonia. If the liver function is compromised, encephalopathy may occur.

6. Predisposition to the development of carcinoma of the colon in 30% of patients after a latent period of 10 years.

Ileal conduit (Fig. 47.10)

A segment of terminal ileum is isolated with intact blood supply. The two ureters are implanted in the ileal segment. At one end the ileal segment is closed, and is fixed at the other end to the skin as an ileostomy. A special appliance is applied to the ileostomy to collect urine. No ascending infection occurs. Hyperchloraemic acidosis may occur but is less the colon diversion.

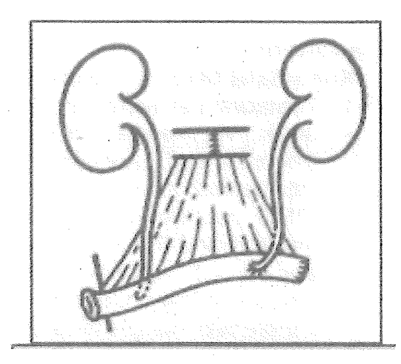


Fig. (47.10): Ileal conduit

Rectal bladder (Fig. 47.11)

The sigmoid colon is divided above the recto-sigmoid junction. The proximal end is brought out as a colostomy. The distal end is closed and the two ureters are implanted in the rectum, which will now work as a urinary bladder.

Advantages

1. Easy and safe operation.
2. The patient will usually be continent for urine.
3. No ascending infection or hyperchloraemic acidosis.



Disadvantages

Presence of the colostomy but the inconvenience of the colostomy is rapidly tolerated by the patient.

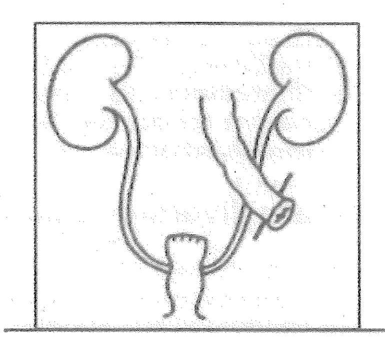


Fig. (47.11): Rectal bladder

Continent urinary diversion

The idea is to separate a segment of bowel with intact blood supply and change its configuration so that it accommodates a large volume for urine. The ureters are implanted in the new bladder, which in turn, is anastomosed to the bladder neck or the urethra. Nowadays, this is the standard urinary diversion after cystectomy as it preserve renal function and is most physiological.

Prostate cancer

Incidence

Carcinoma of the prostate is a common malignant lesion that affects males usually over the age of 65 years.

Predisposing factors

1. Family history. The relative risk is greatly increased if one or more first-degree relatives have the disease (father or brothers).
2. Race. There is a striking difference in racial distribution of prostate cancer. African Americans have the highest incidence of the disease while native Asian populations have the lowest incidence.
3. Age. In autopsy studies, the prevalence of prostate cancer shows a constant rise over successive decades starting at the age of 50 years.

Pathology

Gross picture

The majority of neoplasms (75%) arise from the peripheral zone, beneath the capsule. The tumour appears as hard infiltrating pale nodule.

Microscopic picture

95% of prostatic cancer are adenocarcinoma derived from the acinar epithelium. There is a grading system of prostatic cancer (Gleason's grade) based on the degree of glandular differentiation and growth pattern. Grade 2 is the least malignant while grade 10 is the most malignant.

Spread

- Direct invasion involves the seminal vesicles, periprostatic tissue and rarely the rectum as the fascia of Denonvillier acts as a barrier. Ureteric obstruction occurs in 20% of cases.
- Lymphatic spread is to the intemal, external and common iliac lymph nodes then to the para-aortic lymph nodes. The obturator lymph nodes may be also involved.
- Blood spread. 90% of blood borne metastases occur in bones especially the lower vertebrae, neck of femur, pelvic bones and the ribs. The preference of pelvic and lumbar vertebrae for metastases is attributed to the reversal of blood flow from the vesico-prostatic plexus to the emissary veins of the pelvic bones during coughing or sneezing. Metastases are usually osteoblastic. Rarely metastases develop in the lungs or the liver.

Clinical features

Presentations

1. Symptoms of urinary outflow obstruction. The majority of patients complain of difficult micturition, acute retention, incontinence, haematuria or painful micturition. A short history of these symptoms in a male above 65 years is suggestive.
2. Pathological surprise. The diagnosis may be reached only after routine histopathological examination of a prostate that was removed with a preoperative diagnosis of benign prostatic hyperplasia.
3. Metastases. The first presentation may be due to metastases. Backache, sciatica, oedema of the lower limb, spontaneous fracture or paraplegia may occur.
4. Routine screening with prostate specific antigen (PSA). A high level raises suspicion and warrants further investigations for confirmation of the diagnosis.

Examination

Digital rectal examination reveals a hard, irregular induration in a part or the whole of the gland. The median sulcus is obliterated. It is to be noticed that a normal digital rectal examination does not exclude the diagnosis.

Staging system TNM (Fig. 47.12)

T	T_0	No clinical abnormality.
	T_1	Nodule in one lobe.
	T_2	Diffuse disease.
	T_3	Extension to the seminal vesicles.
	T_4	Lesion is fixed to other tissues.
N	N_0	No evidence of involvement of lymph nodes.
	N_1	Involvement of one regional node.
	N_2	Involvement of several regional nodes.
	N_3	Fixed mass of regional nodes.
	N_4	Involvement of common iliac or para-aortic nodes.
M	M_0	No evidence of distant metastases.
	M_1	Distant metastases.

Investigations

Laboratory

1. **Prostatic specific antigen (PSA)** is the most useful marker in the diagnosis and follow-up. The normal value is 0-4 ng/ml. A raised level suggests prostate cancer. Markedly elevated PSA >20 ng/ml suggests disseminated disease. The serum level is not affected by rectal examination, but it may be mildly raised in non malignant conditions as in prostatitis or benign prostatic hyperplasia.
2. **Acid phosphatase** is raised in 70% of patients with extracapsular or metastatic disease. Acid phosphatase is raised in many other conditions as Gaucher's disease, Paget's disease of bones, and with breast and gastric cancer, After rectal examination the acid phosphatase remains elevated for 24 hours.
3. **Alkaline phosphatase** is raised if there are bony or hepatic metastases.
4. Blood picture and blood urea and creatinine.

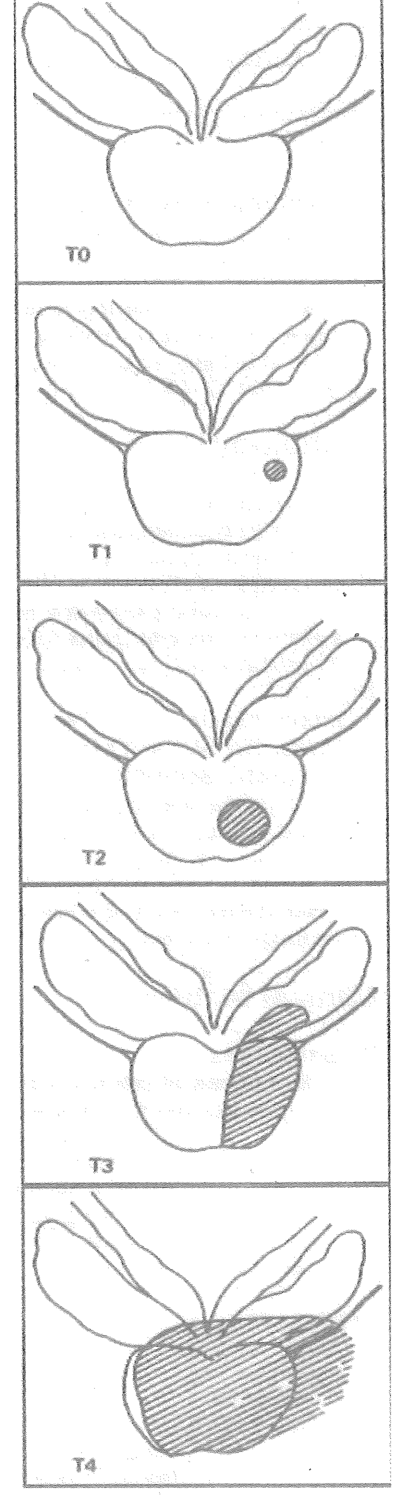


Fig. (47.12): T stage of prostate cancer

Imaging

1. **Plain X-ray** of the chest, pelvis and spine to detect metastases which are usually osteoblastic.
2. **Bone scan** by Technetium^{99m} will detect areas of increased bone activity. Metastases will appear as hot spots. Scanning is not specific and the scan abnormality should be compared with the clinical examination and plain radiography.
3. **CT** scan of the pelvis can help detect pelvic lymph node deposits, but is not accurate.
4. **Trans-rectal ultrasonography** (TRUS) can give an accurate idea about the extent of the lesion.

Biopsy

Any patient with abnormal digital rectal examination and/or an elevated PSA must undergo multiple trans-rectal biopsies under ultrasound guidance (TRUS).

Treatment

There are many policies in the management of prostatic cancer. Treatment will be dictated by the local extent of the disease, the presence of metastases and the general condition of the patient.

1. **Radical prostatectomy**. This is only performed for early localized disease in a fit patient. It entails removal of the prostate and seminal vesicles together with block dissection of obturator, internal and external iliac lymph nodes. The bladder is then anastomosed to the urethra. Complications include urinary incontinence in 5% and impotence in 30% of cases.

2. **Irradiation**. This is indicated for locally advanced disease and in unfit patients. External beam radiation therapy is applied giving a dose of 7000cGy over 5 weeks, Radiation therapy has some side effects; bladder irritation that produces frequency of micturition or urgency, and rectal irritation that produces diarrhoea or bleeding per rectum. Irradiation has also got a useful role for symptomatic bone metastases as it produces dramatic relief of pain.
3. **Transurethral resection (TURP)**. Those who have bladder outflow obstruction and are not fit for major surgery, can have a satisfactory symptomatic relief after TURP.
4. **Hormonal therapy**. This is the mainstay of treatment in advanced cases or in patients who are unwilling or unfit for surgery or radiotherapy. The rationale for hormonal treatment is that prostate cancer is an androgen sensitive tumour. There are different methods for androgen ablation:
    a. Bilateral orchidectomy or orchiectomy. Orchidectomy means excision of the testis with its tunica albuginea together with the epididymis. Orchiectomy means removal of testicular tissue from inside the tunica albuginea in order to avoid the appearance of an empty scrotum. Both operations are effective, easy and safe. Their disadvantages are loss of libido and some erectile dysfunction.
    b. Luteinizing hormone releasing hormone (LHRH) agonist (e.g., Zoladex). The drug causes initial rise of the level of testosterone for 4-6 weeks then a drop to castrate levels. It gives equal results to castration and has the same side effects. It is an expensive drug.
    c. Antiandrogens (Nutilamide). These act by blocking testosterone receptors by competitive inhibition. They are used as adjuvants to castration or LHRH agonists.
    d. Oestrogens act as anti-androgens. A small dose of stilbestrol I mg t.d.s is used. Its major disadvantage is that it predisposes to deep vein thrombosis. Ethinyl oestradiol may also be used.
5. **No treatment**. Small well-differentiated tumours in elderly men may be managed by "watchful waiting", particularly if their anticipated life expectancy is less than 10 years.

Fig. (47.13) is an algorithm for the treatment of prostatic cancer

Prognosis

- The life expectancy of a patient with an incidental finding of focal carcinoma of the prostate is that of the normal population.
- With tumours localized to the prostate, the 10-year survival rate is 50%.
- If metastases are present, the 10-year survival rate drops to 10%.

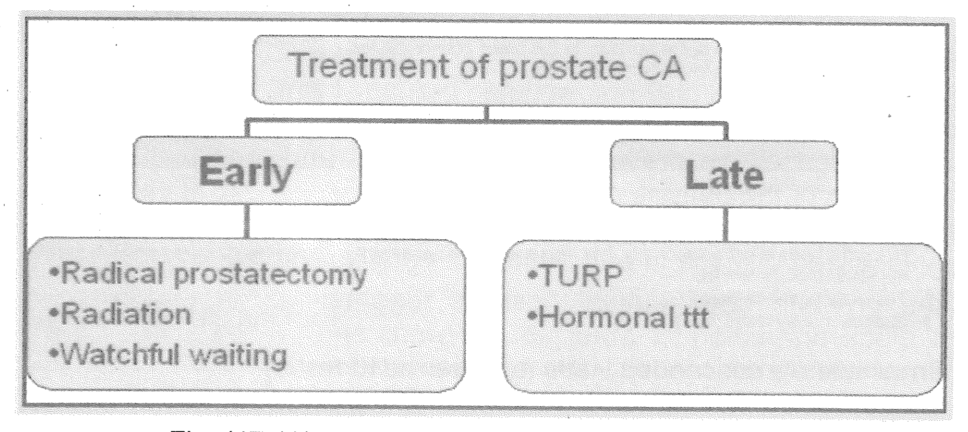


Fig. (47.13): Treatment of prostatic cancer

THE TESTES AND SCROTUM

CHAPTER CONTENTS

Development of the testes

The testis develops from the mesodermic genital fold behind the peritoneum and below the developing kidney. It is attached to the posterior abdominal wall by a mesentery (the mesorchium). It receives its blood vessels and nerves high in the abdomen.

- The testis descends downwards, forwards and medially towards the scrotum pulling with it its blood and nerve supply as well as a fold of peritoneum (the processus vaginalis). The distal portion of the processus vaginalis persists as the tunica vaginalis.
- As the testis reaches the lower part of the abdomen, it unites with some of the mesonephric tubules which give the vasa efferentia. The epididymis and vas deferens develop from the Wolffian duct.
- Normally, the testis reaches the internal inguinal ring at the sixth month, the external ring at the eighth and the scrotum just before birth.
- Descent of the testis depends upon:
    - Differential growth of the body; the cranial segment grows faster than the caudal segment.
    - Maternal chorionic gonadotrophins and pituitary gonadotrophic hormones play a definite role in the development and descent of the testis.
    - Contraction of the gubernaculum, (fibromuscular tissue extending from the bottom of the scrotum to the lower pole of the testis) directs the testis towards the scrotum.

A retractile testis is a normal finding in infants and young children. It requires no treatment

Congenital anomalies of the testes

1. Maldescent of the testis
    a. Incompletely descended testis (undescended testis).
    b. Ectopic testis.
    Retractile testis is not an anomaly. It is discussed here because it is commonly confused with the above- mentioned two anomalies.
2. Testicular agenesis (rare).
3. Supernumerary testis (rare).

Incompletely descended testis (undescended testis)

Incidence

- This disorder affects about 1% of all males and is commoner on the right side.
- It may be bilateral in about 20% of cases. Cryptorchidism means bilateral undescended impalpable testes.
- The incidence is higher in premature infants.

Aetiology

The predisposing factors for incomplete descent are:

1. Mechanical factors leading to failure of the testis to engage in the inguinal canal.
    a. Short mesorchium.
    b. Large size of the testis and epididymis.
    c. Shortness of the spermatic vessels or the vas deferens.
    d. Adhesions fixing the testis in higher places.
    e. Rupture of the gubemaculum.
2. Deficiency of gonadotrophic hormones.

Pathology

Site

- The testis may arrest anhere along the normal line of descent.
- In the abdomen (intra-abdominal).
- In the inguinal canal.
- At the external ring.
- At the neck of the scrotum (high scrotal).

> An undescended testis should be operated upon before the age of two years.

Consequences

1. Depressed spermatogenesis. Effective spermatogenesis requires a temperature less than that of the body core. This is why the testes are normally placed outside the abdomen, in the scrotum. An undescended testis is exposed to body temperature, and hence, spermatogenic cells are affected. Up to two years, the testis remains almost normal, after that, there is retardation of its development due to the higher temperature. At puberty, irreversible destructive changes in the germinal epithelium occur and in bilateral cases sterility is usually the rule. On the other hand, hormonal function of the testis remains normal, so that secondary sexual characters develop normally even in bilateral cases.
2. Associated oblique inguinal hernia is present in more than 90% of cases.

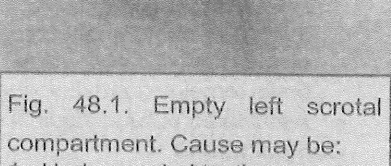


Fig. 48.1. Empty left scrotal compartment. Cause may be:
1. Undescended testis.
2. Ectopic testis.
3. Retractile testis. The scrotal compartment is temporarily empty. You can pull the testis down in place.
4. Testicular agenesis.
5. Testicular atrophy, e.g., after mumps orchitis.

Complications

1. Sterility in bilateral cases.
2. Increased incidence of trauma especially in inguinal canal type.
3. Torsion of the spermatic cord.
4. Increased liability to testicular malignancy especially in intra-abdominal type. This is genetically determined, and may affect the patient even after a successful orchidopexy.
5. Complications of associated hernia.
6. Psychiatric disturbances.

Clinical features

Symptoms

Absence of one or both testes is usually discovered by the mother or the attending doctor. The condition is present since birth.

Signs

1. The ipsilateral side of the scrotum is empty (Fig. 48.1) and underdeveloped.

2. The testis is palpable if it lies high in the scrotum. A testis that lies in the inguinal canal may be felt with difficulty. The testis is identified by the patient as it gives a characteristic sickening sensation when compressed.
3. An associated hemia may be present.

Differential diagnosis

1. Ectopic testis (see later).
2. Retractile testis (see later).
3. Testicular agenesis.
4. Atrophy of the testis, e.g., after mumps orchitis.
5. Hermaphroditism should be excluded in bilateral cases.

Investigations

1. Hormonal assay (LH and testosterone hormone) to exclude cases of anorchia in bilateral cases.
2. Ultrasonography and CT scan can help to localize the site of the testis when it is not felt.
3. Laparoscopy detects an intra-abdominal testis.

Treatment

Surgery is the standard treatment. **Orchidopexy** should be performed before the age of two years.

Principles of surgery

1. Excision of a concomitant hernia sac (hemiotomy).
2. Division of adhesions to ensure that the vas deferens and the testicular vessels have enough length to reach the bottom of the scrotum.
3. Fixation and retaining the mobilized testis in the scrotum.

Steps

1. Lower abdominal crease incision.
2. The inguinal canal may be opened if the testis is not found in the superficial inguinal pouch.
3. The cord and the testis are mobilized and freed from the surrounding structures.
4. An associated hernia sac is dissected and excised at the internal ring.
5. If extra-length is still needed, the vessels are dissected from the peritoneum and the inferior epigastric artery is divided to abolish the angulation of the vas around it.
6. The empty half of the scrotum is stretched to prepare a scrotal bed for the testis.
7. The mobilized testis is fixed in the scrotum to avoid retraction. The most popular method for fixation of the testis is to place it in a Dartos pouch created between the skin of the scrotum and the Dartos muscle.

Management of difficult cases

The difficulty usually arises from a short testicular artery. This can be dealt with by one of the following methods:

- Staged orchidopexy. The testis is brought down in more than one stage.
- Microvascular technique. Division and reanastomosis of the testicular vessels at a lower level to the inferior epigastric vessels.
- Orchidectomy is done only if the other testis is normal and the undescended testis is atrophic.

Ectopic testis

The testis has already passed out of the external inguinal ring. It then deviated outside its normal route to a subcutaneous ectopic position.

Aetiology

The condition may be caused by the pull of an accessory gubernaculum.

Pathology

The hormonal and the spermatogenic functions are normal. The possible sites of an ectopic testis are shown in Fig. 48.2 (a) the inguinal region, (b) in the perineum, (c) at the root of the penis or (d) in the femoral triangle.

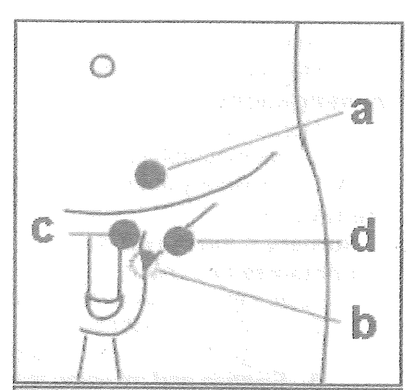


Fig. 48.2. Possible sites of an ectopic testis.
a. Inguinal (subcutaneous).
b. Perineum.
c. At root of penis
d. Femoral.

Clinical features

- Empty scrotal compartment since birth.
- The testis is felt in one of the above-mentioned ectopic sites.
- The most frequent position is the superficial inguinal pouch. Being subcutaneous, the testis is easily felt and bulges more if the muscles contract while an undescended testis disappears because it is usually deep in the inguinal canal.

Treatment

Surgical replacement of the testis in the corresponding scrotal compartment.

Retractile testis

A retractile testis is not a disease because the testis has descended normally to the bottom of the scrotum. It is commonly seen in young children due to exaggerated cremasteric reflex where the testis is pulled up due to contraction of the cremasteric muscle. The testis lies in the superficial inguinal pouch. It can be differentiated from incompletely descended testis by:

1. The scrotum is well developed.
2. The testis can be pushed into the scrotum by sliding the fingers down the inguinal canal, especially if the child squats and draws the thigh to the abdomen, or if the child is examined during sleep in a warm room. A retractile testis is diagnosed if it can be brought down to touch the bottom of the scrotum at any time.

Treatment

No treatment is required. Reassure the parents that the condition will be cured spontaneously. As the testis becomes heavier, the cremasteric muscle can no longer pull it upwards.

Torsion of the spermatic cord (torsion of the testis)

Testicular torsion is a surgical emergency. It means torsion of the testis and epididymis around the axis of the spermatic cord.

Aetiology

This is an uncommon problem occurring usually in adolescents. **Predisposing causes** include:

1. Imperfect descent of the testis (incomplete or ectopic).
2. Long mesorchium.
3. Capacious tunica vaginalis that surrounds the whole testis. The testis is suspended loosely inside its cavity.

The **exciting cause** is usually a minor trauma or straining at defecation, but spontaneous torsion sometimes occurs.

Pathology

1. The testis rotates from outside inwards (Fig. 48.3). It becomes oedematous and congested. The colour becomes dark and finally gangrene occurs within 8-12 hours if the condition is not treated.
2. The spermatic cord shows the twists (one or more). If torsion persists, the blood vessels are thrombosed.
3. There is hydrohaematocele in the tunica vaginalis.
4. The scrotal skin is red and oedematous.



Fig. 48.3. Direction of torsion.

Clinical features

Symptoms

- Sudden severe agonizing pain in the scrotum, groin and the lower abdomen.
- It may be associated with vomiting (not persistent).

Signs

1. The scrotum is swollen with irreducible acutely tender swelling. The overlying skin is oedematous and red (Fig. 48.4). The testis is higher than normal.
2. Shock with tachycardia and sweating may be present.

Testicular torsion is a surgical emergency. Even in case of doubt urgent surgery is still recommended.

Differential diagnosis

1. Acute epididymo-orchitis. Table 48.1 illustrates the differences between torsion of the testis and acute epididymoorchitis.
2. Torsion of an undescended testis has to be differentiated from a strangulated inguinal hernia.



Investigations

1. Urgent Doppler to detect obstruction of the flow in the vessels.
2. Urgent Duplex scan, if available, adds more confirmation by detecting the thrombus in the vessels as well.
3. Exploration. If the previously-mentioned investigations are not available and the diagnosis is suspected, exploration is indicated even if it proves negative. Any delay may lead to loss of the testis.

Fig. 48.4. Torsion of left testis. A painful swollen red scrotum is easily confused with acute epididymo-orchitis.

Treatment

Urgent surgery

1. Scrotal incision.
2. The torsion is undone.
3. If the testis is viable, the tunica vaginalis is everted and the testis is anchored to the bottom of the scrotum to prevent recurrence.
4. If the testis is gangrenous (Fig. 48.5), a high orchidectomy; above the level of twists; is done.
5. The opposite testis should be fixed at the same time as the predisposing anomaly is commonly bilateral.



Fig. 48.5. At operation this testis was found gangrenous.

Table 48.1. Differences between torsion of testis and acute epididymo-orchitis.

	Torsion of testis	Acute epididymo-orchitis
Age	Usually adolescents and children	Usually adults or elderly
History	Sometimes mild trauma	Usually UTI symptoms
Temperature	Normal or slightly elevated	Elevated
Elevation of scrotum	Does not alleviate pain	Partial pain relief
Urgent urine analysis	Free	May show pus cells
Urgent Doppler or Duplex	Obstructed testicular vessels	Patent testicular vessels

Inflammatory conditions of the testis and spermatic cord

Inflammation of the testis alone (orchitis) is rare. It is usually associated with inflammation of the epididymis (epididymo-orchitis) or with the sperniatic cord and the epididymis (funiculo-epididymo-orchitis). It may be acute or chronic.

Acute non-specific epididymo-orchitis

This is a fairly common disease.

Mode of infection

1. Ascending infection (common route) through the lumen of the vas secondary to urethritis, prostatitis and seminal vesiculitis. It may occur spontaneously, or after instrumentation (urethral catheterization or cystoscopy). The commonest organisms are gonococci, E. coli, proteus, streptococci, staphylococci and Chlamydia.
2. Blood borne infection (rare) secondary to specific fever as mumps.

Clinical features

1. There may be history of prostatitis or mumps.
2. Acute severe scrotal and groin pain.
3. Constitutional symptoms (malaise, fever, rigors, etc.) may be present.
4. The epididymis and the testis are swollen, tender with redness and oedema of the skin. A small secondary hydrocele may be present.
5. Inflammation may subside spontaneously, but recurrence is common.
6. Commonly pus cells are detected in urine analysis.

Treatment

1. An antibiotic, usually a member of the quinolones group, is given for 2-3 weeks. A residual epididymal mass may persist for up to 6 weeks and then resolves spontaneously.
2. Rest for the first few days.

Chronic specific epididymo-orchitis

Tuberculosis

- The epididymis is the primary site of affection in genital tuberculosis.
- The disease occurs mainly in young males.
- Infection usually reaches the epididymis through the vas deferens from tuberculosis of the seminal vesicle and the prostate.
- Multiple tubercles affect the tail of epididymis. The nodules enlarge and may undergo caseation and cold abscess formation. It becomes adherent to the skin and bursts to form a tuberculous sinus on the posterior aspect of the scrotum.
- The vas deferens is thickened and is studded with multiple tubercles (beading).
- The testis usually remains free for a long time, but in neglected cases it may be affected.
- Treatment is essentially medical by anti-tuberculous drugs. If there is no response to conservative treatment after two months excision of the vas deferens and epididymis is indicated.

Filariasis

- Chronic funiculo-epididymo-orchitis may follow acute funiculo-epididymitis or is chronic from the start.
- As the disease affects the lymphatics, the spermatic cord is matted, i.e., the vas cannot be differentiated from the other constituents of the cord.
- There are firm to hard masses in the cord, but breaking down does not occur.
- There may be secondary hydrocele.

Bilharziasis

- Bilharzial affection of the cord or testis can occur as a complication of urinary or intestinal disease. It is a chronic disease from the start.
- The disease affects the lower part of the cord and the epididymis.
- The cord is thickened but is not matted. It contains Bilharzial masses that vary greatly in size.
- The testis and the tunica vaginalis are usually normal.

Neoplasms of the testis

Practically 99% of testicular neoplasms are malignant. They constitute 1-2% of malignant tumours in males and occur at a relatively young age.

Aetiology

Incompletely descended testis, especially the intra-abdominal variety. The incidence of malignancy in undescended testis is 15 times the normal population. 10% of patients with testicular tumours have history of undescended testis. It is to be noticed that surgery for an undescended testis to bring it to the scrotum does not reduce the high susceptibility to malignancy.

Pathology

Classification

1. Germ cell tumours
   a. Seminoma 40%
   b. Non-seminomatous germ cell tumours (teratomas) 32%
   c. Combined seminoma and teratoma 14%
2. Interstitial tumours (rare)
   a. Leydig cell tumour
   b. Sertoli cell tumour
3. Lymphoma (rare)

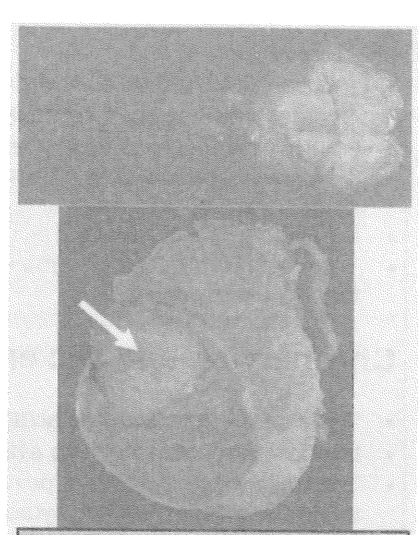


Fig. 48.6. Seminomas. Notice that cut sections look homogenous.

Seminoma

- This is the commonest neoplasm of the testis. It occurs between 30-50 years of age.
- Macroscopically (Fig. 48.6), the testis is large, firm and smooth. On section it is homogenous and pink creamy in colour. Sometimes, it is lobulated due to the presence of fibrous septa. Necrosis may occur in rapidly growing tumours.
- Histologically, seminoma is derived from the seminiferous tubules, so the cells resemble spermatocytes. They are rounded or oval with clear cytoplasm and large rounded nuclei. The cells are arranged in sheets separated by fibrous tissue stroma, which may be infiltrated with lymphocytes. Seminoma disseminates mainly by lymphatics and rarely by blood.

Teratomas

- The tumour occurs between the ages of 20-35 years. It arises from totipotent cells which can differentiate into the three embryonic layers (ectoderm, mesoderm and endoderm). Usually one of them predominates and turns malignant.
- Macroscopically (Fig. 48.7), the tumour varies in size. The shape of the testis is retained, but with irregular surface. The cut section may show cysts that contain gelatinous material. It looks heterogenous and there may be areas of haemorrhage and necrosis.
- Histologically teratomas are classified as following:
  - Teratoma differentiated. There are no histologically recognizable malignant components, but it can metastasize, so it is not benign. The best example is dermoid cyst.
  - Malignant teratoma intermediate (commonest testicular teratoma).
  - Malignant teratoma anaplastica. The neoplasm is composed of undifferentiated embryonal cells of yolk sac origin. These cells produce alpha-fetoprotein.
  - Malignant teratoma trophoplastica. This was previously known as choriocarcinoma. It is the most malignant tumour known.

Fig. 48.7. Nonseminomatous germ cell tumours (teratomas). Notice heterogenicity and cystic spaces on cut section.

Interstitial cell tumours

The tumour arises early in life either in the cells of Leydig or those of Sertoli.

- Leydig cell tumours usually occur before puberty. They produce excessive amounts of androgens, and lead to infant Hercules (sexual precocity and extreme muscular development).
- Sertoli cell tumours occur after puberty and produce oestrogens leading to gynaecomastia, loss of libido and aspermia.
  Pregnancy test is positive.

Clinical features

In 10% of cases there is history of trauma that merely attracts the patient's attention to the presence of a swelling

1. **Typical cases.** The first symptom is painless enlargement of the testis with sense of heaviness. Pain is present in 30% of the cases. On examination, the testis is enlarged, hard, smooth and heavy. Later, soft bossy areas appear. Testicular sensation is lost early in the course of the disease. Lax secondary hydrocele is present in 10% of cases. The epididymis is at first normal, then becomes stretched over the swelling and finally becomes infiltrated with the tumour. The para-aortic lymph nodes may be palpable just above the umbilicus on either side. The inguinal lymph nodes are not affected unless the scrotal skin is infiltrated. The liver may be palpable.
2. In some patients the testicular enlargement is not noticed by the patient who complains of symptoms due to either lymphatic or blood metastases, e.g., epigastric pain or cough, dyspnea and haemoptysis. The supraclavicular nodes, particularly on the left side may be enlarged by metastases.
3. An abdominal mass with an empty scrotal compartment should raise the suspicion of malignant transformation in an abdominal undescended testis.
4. Hormonal effects
   a. Infant Hercules.
   b. Feminising tumours.
5. Atypical presentations
   a. Some cases may simulate epididymo-orchitis. Acute pain and swelling may be due to haemorrhage.
   b. Hurricane type. Fatal termination occurs due to metastases from highly malignant tumours.
   c. Slowly growing type which takes 2-3 years to develop.

Staging

Stage I	Tumour in the testis only.
Stage II	Involvement of lymph nodes below the diaphragm.
Stage III	Involvement of lymph nodes above the diaphragm.
Stage IV	Systemic metastases.

Investigations

Whenever a testicular neoplasm is suspected the following investigations are performed:

1. **Tumour markers**
   a. Beta fraction of human chorionic gonadotrophin is raised in 100% of patients with choriocarcinoma and in less than 10% in seminoma. Sustained elevation of tumour markers after treatment suggests residual disease.

b. Alpha fetoprotein (AFP) may be elevated in non-seminomatous germ cell tumours (teratomas) but never in seminomas.
c. Lactic dehydrogenase.

2. Scrotal ultrasound, can confirm the presence of the testicular tumour and differentiate it from other lesions, e.g., epididymitis.
3. Chest X-ray.
4. Abdominal CT scan for lymph nodes and liver deposits.
5. Biopsy and frozen section examination are done during operation. Biopsy should never be taken through the scrotum neither by incision nor by needle aspiration.

Treatment

Initial treatment is by high orchidectomy

Through an inguinal incision the spermatic cord is identified and isolated at the internal inguinal ring. A vascular clamp is applied as high as possible on the spermatic cord to avoid the risk of blood dissemination while manipulating the tumour. The testis can now be delivered out and is inspected. If there is a frank testicular tumour the testis and the spermatic cord are excised after doubly ligating and dividing the latter at the internal ring. If the surgeon is in doubt, biopsy is taken for frozen section examination while the clamp is in place. A scrotal incision is contraindicated as it opens a way for spread to inguinal lymph nodes.



Fig. 48.8. High orchidectomy.
Notice:
1. The use of an inguinal incision.
2. Cord is clamped high at internal ring.

Never puncture nor incise the scrotum in a case that is suspected to be testicular cancer. This opens a way for spread to inguinal nodes.

Further management depends on the type and stage of the disease.

- **Seminoma**. The tumour is radiosensitive and, therefore, radiotherapy is relied upon when the disease is fairly limited. With advanced disease, more reliance is made on chemotherapy because of its systemic value.
    - Stage I — Radiotherapy for para-aortic lymph nodes.
    - Stage II — Radiotherapy that extends above the diaphragm.
    - Stage III — Chemotherapy with or without radiotherapy.
    - Stage IV — Chemotherapy.
- **Teratoma**. The tumour is less sensitive to irradiation than a seminoma. However, the introduction of combination chemotherapy based on cisplatin has greatly improved its outcome.
    - **Stage I**. Retroperitoneal lymphadenectomy is practiced in the USA. In some other countries an expectant policy is adopted, and surgery or chemotherapy is done only if the patient develops lymph node enlargement.
    - **Stage II-IV**. Combination chemotherapy.

  Success of therapy is known by reduction of tumour markers to normal levels. Incomplete reduction indicates residual tumour. Re- elevation of the tumour marker level in the follow-up period signifies recurrence.

Varicocele

Varicocele is elongation, dilatation and tortuosity of the pampiniform plexus of veins, i.e., varicose veins of the spermatic cord (Fig. 48.9).

Surgical anatomy

- The veins of the testis and epididymis form the pampiniform plexus. It is formed of 4-8 veins in the inguinal canal. In the retroperitoneal area, they are reduced to 1-2 testicular veins.
- On the right side, the testicular vein ends in the IVC while the left empties in the left renal vein.
- The pampiniform plexus and the testicular veins are devoid of valves except at their termination.

Varicocele is usually primary and rarely secondary.

Primary varicocele

Aetiology

Primary varicocele usually affects young adults (15-30 years). Its exact cause is not known but may be due to congenital weakness of venous walls, which may be due to familial weakness of mesenchyme (associated with hernia, varicose veins, and flat feet).

Primary varicocele almost always affects the left side. The right side is rarely affected. Sometimes it is bilateral. The tendency for the left side may be due to:

1. The right angled termination of the left spermatic vein into the left renal vein impedes the venous return, in contrast to the oblique termination of the right vein into the IVC.
2. The lower position of the left testis with longer and more dependent plexus.
3. Compression of the left testicular vein by the heavily loaded pelvic colon in constipated patients.

Fig. 48.9. Varicocele.

Complications

Hypofertility. Varicocele, particularly when bilateral can reduce the sperm count or vitality and thus reduce fertility. This is presumed to be the result of the higher temperature in the scrotum produced by venous congestion, which affects spermatogenesis.

Clinical features

Symptoms

1. Usually, the condition is symptomless and is discovered at a medical check-up.
2. Dragging sensation or aching pain in the testis especially on prolonged standing or in hot weather.
3. The patient may complain of infertility.

Signs

1. The affected side of the scrotum hangs lower than normal.
2. The varicocele cannot be felt while the patient is lying down. On standing the dilated veins are felt above the testis, and are described to feel like a 'bag of worms'. Large varicoceles are also visible.
3. Varicosities may be present in the scrotal skin.
4. The varicocele forms a soft compressible inguinoscrotal swelling which gives a fluid thrill on cough.

Investigations

1. Doppler or duplex scan can detect reversal of blood flow in the testicular vein.
2. Semen analysis for cases of infertility.
3. Abdominal ultrasound to exclude cases of secondary varicocele, e.g.. those due to hypernephroma.

Treatment

Conservative treatment

- In early cases, the patient is reassured.
- For pain a scrotal suspender or close-fitting underpants can be used especially in hot weather.

Surgical treatment

Indications

1. Deficient spermatogenesis (oligospermia). Sperm count and infertility improve in 30-50% of cases after surgery.
2. A large painful varicocele, when the pain does not respond to conservative measures.

Operations

Prevention of venous reflux is achieved by attacking the venous return of the testis at one of these levels:

1. Ligation and division of the testicular vein(s) in the extraperitoneal space as it emerges from the internal ring (Palomo's operation).
2. Ligation and division of the pampiniform plexus in the inguinal canal.
3. Ligation and division of the pampiniform plexus high in the scrotum.

Secondary varicocele

This rare condition is due to obstruction of the venous flow in the spermatic vein by an abdominal tumour, usually hypernephroma. The obstruction is produced by:

- Intramural growth of the tumour in the renal vein or inferior vena cava impeding venous drainage.
- Pressure from outside by the renal mass.

Clinical features

Secondary varicocele differs from the primary type:

1. It occurs after the age of 40 years.
2. It affects both sides equally.
3. It develops rapidly and enlarges in a few weeks.
4. It does not disappear on lying down or if the scrotum is elevated.

Treatment is that of the cause.

Epididymal cysts and spermatocele

Pathology

Cysts of the epididymis are of unknown aetiology. They often arise from the head of the organ and are filled with clear fluid (Fig. 48.10).

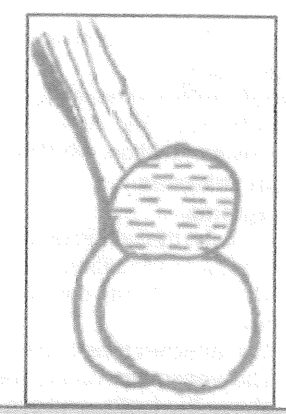


Fig. 48. 10. Epididymal cyst

Occasionally the cyst contains spermatozoa which make the fluid turbid. Such a cyst is called a "spermatocele'.

Clinical features

Symptoms

The patient complains of a painless scrotal swelling. The cyst is usually small, yet it sometimes attains such a large size that it looks to the patient like a third testis.

Signs

- On examination the swelling is found to be purely scrotal, lying just above and slightly behind the testis.
- Epididymal cysts are nontender, cystic, and, with the exception of spermatoceles, are brilliantly translucent.
- The main difference from a vaginal hydrocele is that an epididymal cyst is felt separate from the testis.

Treatment

- Most epididymal cysts deserve no treatment.
- Exceptional large, or rapidly growing cysts are surgically excised. Utmost care should be taken not to injure the epididymis while removing the cyst.

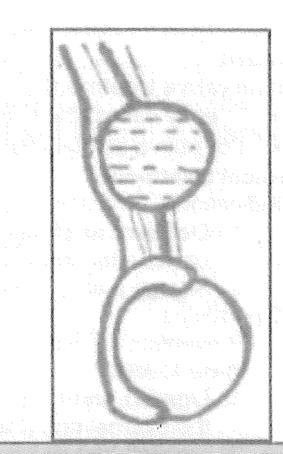


Fig. 48.11. Encysted hydrocele of the spermatic cord.

Hydrocele

A hydrocele is a collection of clear serous fluid inside a part of the processus vaginalis.

Classification

1. **Hydrocele of the spermatic cord**
   - Encysted.
   - Hydrocele of a hernia sac.
2. **Hydrocele of the tunica vaginalis**
   - Congenital.
   - Infantile.
   - Vaginal, primary or secondary.

Hydrocele of the spermatic cord

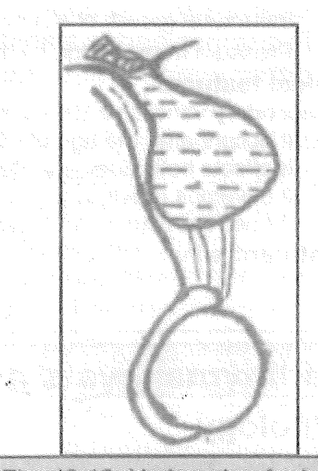


Fig. 48.12. Hydrocele of a hernia sac.

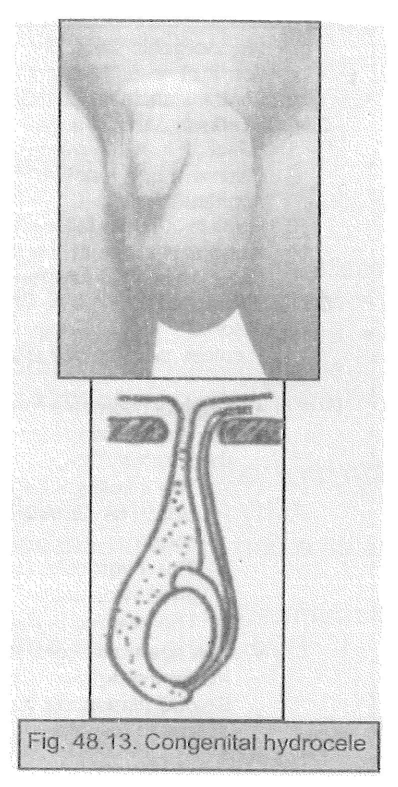
Encysted hydrocele of the spermatic cord (Fig. 48.11)

- **Origin**. The condition is due to persistence of the intermediate part of the processus vaginalis. Although congenital, it usually appears in childhood because fluid takes time to accumulate.
- **Clinical features.** Encysted hydrocele of the cord forms a small, tense, translucent, cystic swelling in the scrotal part of the spermatic cord, usually at the neck of the scrotum. It is oval in shape with the long axis along the cord. The swelling is slowly growing, and is neither painful nor tender. It can be moved from side to side but not from above downwards. The cyst is anchored to the testis by a fibrous band,

representing the obliterated portion of the processus vaginalis, with gentle traction on the testis, it is pulled downwards and becomes fixed, i.e. loses its normal mobility in the transverse axis across the spermatic cord.
- Treatment is excision through an inguinal incision.

Hydrocele of canal of nuck is a similar condition that occurs in a female in relation to the round ligament. The hydrocele is wholly or partially in the inguinal canal. Treatment is the same.

Hydrocele of a hernia sac (Fig. 48.12)
- This swelling is caused by fluid distension of an empty hernia sac which has been shut off from the peritoneal cavity by omentum or by adhesions.
- **Clinical features**. There is history of a hernia. Later, there is an irreducible pyriform translucent cystic swelling in the upper part of the cord. The swelling is mobile from side to side but not from above downwards. Gentle traction on the testis does not alter the position or the mobility of the swelling.
- Treatment is that of the hernia.


Fig. 48.13. Congenital hydrocele

Hydrocele of the tunica vaginalis
Congenital hydrocele (Fig. 48.13)

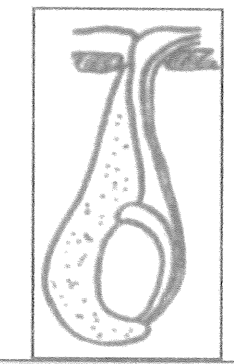
This variety is due to persistence of the whole processus vaginalis. The communication with the peritoneal cavity is too small to permit the development of a hernia. It forms a cystic transluent, inguinoscrotal swelling. The hydrocele is intermiffent; it fills gradually on standing and empties very slowly if the patient lies down and the scrotum is elevated.

Treatment

Operation. Through a lower abdominal crease incision, the sac is divided into two parts. The upper part is transfixed at the level of the internal ring as a hernia and the lower part is either everted, or is just left undisturbed.

Infantile hydrocele (Fig. 48.14)

Infantile hydrocele is the result of incomplete obliteration of the processus vaginalis, so the tunica extends up to the internal ring, but does not communicate with the peritoneal cavity. The differentiation from a congenital hydrocele is known only at operation. Treatment is by eversion of the tunica.


Fig. 48.14. Infantile hydrocele.

Vaginal hydrocele (Fig. 48.15)

This is a fluid collection in the tunica vaginalis which may be idiopathic (primary hydrocele) or may be due to an underlying lesion (secondary hydrocele).

Secondary hydrocele is uncommon. It is secondary to a disease of the cord or the testis. This disease may be acute (epididymoorchitis) or chronic (malignant or chronic inflammation). It is usually small and soft. With testicular cancer the fluid may be bloodstained. Treatment is that of the cause.

Primary vaginal hydrocele is the most frequent type of hydrocele. The following description applies to this primary type.

Aetiology

Primary vaginal hydrocele occurs in middle aged and elderly males. The cause of this condition is not known. Chronic congestion and irritation by repeated trauma, and/or diminished fluid absorption by the tunica vaginalis are possible causes.

Fig. 48.15. Vaginal hydrocele.

Pathology

- There is accumulation of fluid in the tunica vaginalis, which may reach an enormous size.
- It may be unilateral or bilateral.
- The tunica becomes thickened, fibrosed and rarely calcified.
- The hydrocele fluid is amber yellow, thin and contains water, inorganic salts, fibrinogen and albumin in a concentration that resembles a transudate. In old standing cases, it contains cholesterol.



Complications are infrequent

1. Infection usually after aspiration leads to pyocele.
2. Haemorrhage leads to haematocele either after trauma, or spontaneously.
3. Rupture, usually follows trauma.
4. Huge expansion of the scrotum leading to in drawing of the penis which may interfere with micturition and intercourse.

Clinical features

Symptoms

Painless swelling of the scrotum.

Signs

- Vaginal hydrocele forms a purely scrotal swelling, i.e., the fingers can reach above the swelling at the neck of the scrotum.
    - The swelling is pyriform, cystic and dull on percussion. The surface is smooth. There is no impulse on cough.
    - The hydrocele fluid surrounds the testis, which is often impalpable.
    - The swelling is translucent on transillumination.
- The spermatic cord, epididymis and the testis should be examined to exclude cases of secondary hydrocele. Scrotal ultrasound helps examination of a testis that is difficult to palpate.

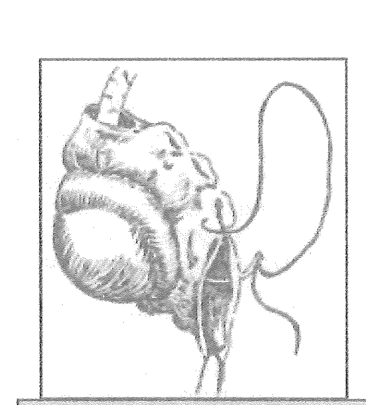


Fig. 48.16. Eversion of tunica vaginalis.

Treatment

Treatment is essentially surgical. Aspiration is not recommended because recurrence is inevitable. Furthermore it may be complicated by infection or haemorrhage.

Surgical options

1. Eversion of the tunica vaingalis (Fig. 48.16). A scrotal incision is done under general or spinal anaesthesia. The tunica vaginalis is opened and evacuated. The cut

edges are everted behind the testis and are sutured behind the epididymis by continuous sutures.

2. **Excision of the tunica vaginalis** (Fig. 48.17) is indicated for a large hydrocele or for thick walled or calcified tunica vaginalis.
3. **Lord's operation** (Fig. 48.18). This operation is suitable for cases where the tunica is not thickened. A small scrotal incision is made through all layers including the tunica. The testis is allowed to prolapse through the wound. The tunica is plicated by a series of radial catgut around the testis. Each stitch extends from the cut end of the tunica vaginalis to the edge of the testis. As there is no tissue dissection, the incidence of postoperative haematoma is much less than with the other two operations.

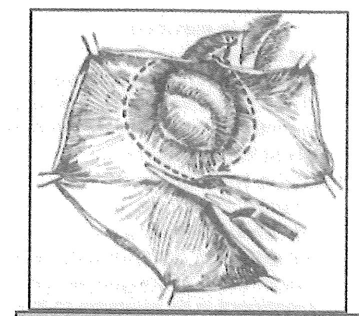


Fig. 48.17. Excision of tunica vaginalis.

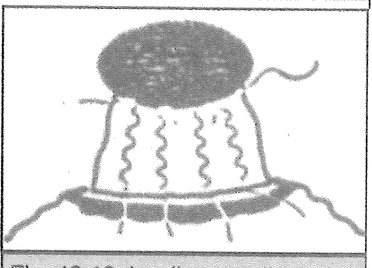


Fig. 48.18. Lord's operation.

Haematocele, pyocele and chylocele

Haematocele

Haematocele is accumulation of blood or blood-stained fluid in the tunica vaginalis. It may be acute or chronic.

Acute haematocele (recent haematocele)

Causes

1. Trauma to the testis, injuring the tunica albuginea.
2. Postoperative, e.g., testicular biopsy.
3. Aspiration of hydrocele.
4. Acute funiculo-epididymo-orchitis.
5. Torsion of the testis.

Clinical features

Blood accumulates rapidly. The tunica becomes tense, tender and opaque with transillumination.

Treatment is surgical for evacuation of the blood and excision of the tunica vaginalis. If there is a tear in the tunica albuginea, it should be repaired.

Chronic haematocele (old clotted haematocele)

Causes

1. Neglected acute haematocele.
2. Malignant neoplasm of the testis.
3. Repeated haemorrhage in blood diseases.

The blood clots. Fibrosis and thickening of the tunica will result.

Complications

1. Infection.
2. Compression and atrophy of the testis.
3. Calcification may occur in old-standing cases and may be mistaken for malignancy.

Clinical features

There is pain and heaviness in the scrotum, together with a swelling which is soft (early) or hard (late). The swelling is opaque on transillumination. The contents of the scrotum cannot be differentiated.

Treatment

1. In early cases, dissection of the clot from the testis and excision of the tunica can be done.
2. In late cases, orchidectomy is performed to exclude malignancy. It is difficult to differentiate between both cases unless the testis is bisected and biopsied.

Pyocele

A pyocele is a collection of pus in the tunica vaginalis.

Causes

1. Infected haematocele.
2. Infected hydrocele especially after tapping.
3. Secondary to suppurative funiculo-epididymo-orchitis.
4. Post-operative.

Clinical features

1. General constitutional manifestations as fever, tachycardia, anorexia, and malaise are present.
2. Locally, there is redness, hotness, oedema and tenderness.

Treatment

1. Incision and drainage of pus.
2. Rest, elevation of the scrotum, antibiotics and antipyretics.

Chylocele

Chyle (lymph) accumulates in the tunica due to rupture of distal lymphatic vessels in case of filariasis. This forms a swelling which is similar to hydrocele, but is opaque on transillumination.

Diseases of the penis

Phimosis

Phimosis means narrowing of the preputial orifice (Fig. 48.19).

Fig. 48.19. Phimosis.

Aetiology

1. Congenital.
2. Acquired due to repeated attacks of inflammations of the prepuce (posthitis).

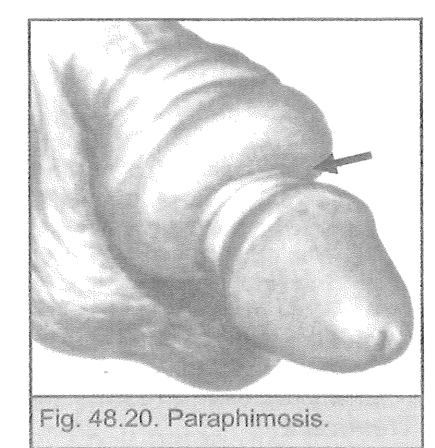


Fig. 48.20. Paraphimosis.

Complications

1. Difficult micturition and straining with back pressure on the kidneys in neglected cases.
2. Infections of the glans penis and prepuce (balanoposthitis).
3. Strangulation of the glans from forcible retraction (paraphimosis, Fig. 48.20).
4. Straining which leads to hernia and rectal prolapse.

Treatment is by circumcision.

Circumcision

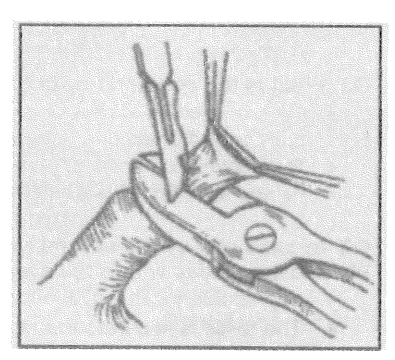
Indications

- In infants
    - On request by the parents for religious or ritual reasons.
    - Recurrent balanitis (inflammation of the glans).
    - Phimosis.
    - Paraphimosis.
- In adults
    - Recurrent balanitis.
    - Failure of prepuce to retract.
    - Prior to radiotherapy for penile cancer.

Methods

Crush excision

This method is used for infants under local anaesthesia.

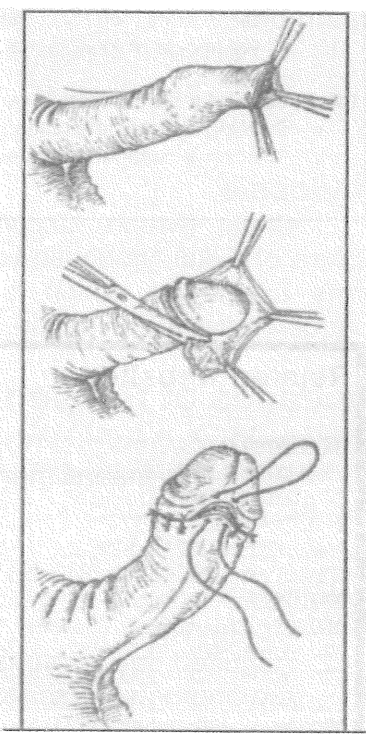

Fig. (48.21): Circumcision by bone cutting forceps

Timing

The first two weeks of life are better avoided because of the bleeding tendency in the newborn. It is recommended to do the circumcision as early as possible after these first two weeks. An alternative is to do it in the first day of life when the neonate still has enough coagulation factors from his mother.

Steps

- Ring infiltration anaesthesia with a local anaesthetic.
- The preputial orifice is stretched and the prepuce is retracted backwards to clean the coronal sulcus from smegma.
- The prepuce is reduced to its normal position. Two artery forceps are applied to the tip of the prepuce which is pulled gently forwards.
- While protecting the glans, a bone-cutting forceps is applied on the prepuce distal to the glans.
- The prepuce is cut with a sharp knife distal to the bone-cutting forceps (Fig. 48.21).
- Usually, no sutures or ligatures are required except if there is bleeding.


Fig. (48.22): Circumcision by dissection

Dissection excision (Fig. 48.22)

- This method is used for children and adults under local or general anaesthesia.
- Retraction of prepuce and cleaning of smegma.
- Three artery forceps are applied to the ventral and lateral aspects of the prepuce.
- The prepuce is slit open in the dorsal midline.
- When the slit reaches the base of the corona, each flap is excised around the penis.
- The skin of the penis is retracted and the vessels are ligated.
- The skin of the penis and the inner layer of the prepuce are sutured with interrupted catgut stitches.

- For adults inhibition of erection for two weeks after the operation is recommended to prevent the sutures from cutting through the skin edges. Bromides or stilbsterol are often used for this purpose.

Complications

1. Bleeding is the most frequent complication. It is sometimes the earliest warning for the affection of the baby with haemophilia. In most cases re-operation is necessary to ligate the bleeding vessels. A bleeding fraenal vessel is controlled by a horizontal mattress suture. Bleeding in a haemophilic is a real problem. Temporary control of haemorrhage by compression should be followed by administration of anti-haemophilic globulin.
2. Removal of too much skin or too little skin. These are avoided by marking the site of cutting before pulling the skin forwards.
3. Injury of the glans penis and external meatus, leading later on to stricture.

Clinical diagnosis of a scrotal swelling

For a clinical diagnosis of a scrotal swelling 4 questions are to be answered:

Q1. Can the examiner's fingers meet above the swelling?

- If the examiner cannot get above the swelling it is an inguinoscrotal swelling. Think of oblique inguinal hernia that reaches down to the scrotum or a varicocele.
    - A hernia gives an expansile impulse on cough and is usually reducible. Reduction may be accompanied by a gurgling sound.
    - A varicocele feels like a bag of worms, is compressible, disappears on lying down and produces a palpable thrill on cough.
    - If the examiner can get above the swelling it is described as a purely scrotal swelling. Proceed to Q2.

Q2. What is the consistency of the purely scrotal swelling?

- Cystic swelling. Think of vaginal hydrocele, epididymal cyst, encysted hydrocele of the spermatic cord or hydrocele of a hernia sac. Proceed to Q3.
- Solid swelling. Think of testicular cancer, old clotted haematocele, chronic epididymitis (tuberculous or non-specific). Proceed to Q4.

Q3. In case of a cystic scrotal swelling is the testis felt separate from the swelling, or is it impalpable because it lies within the swelling?

- Impalpable testis. Think of vaginal hydrocele and confirm it by transillumination.
- The testis is separate from the swelling
    - Epididymal cyst. The swelling is low in the scrotum and is attached to the epididymis. It cannot be moved separate from the testis.
    - Encysted hydrocele of the cord can be moved separate from the testis. Downward traction on the testis limits mobility of the swelling.
    - Hydrocele of a hernia sac is like the former but traction on the testis does not affect mobility.

Q4. In case of a hard scrotal swelling, does it include the testis or is it confined to the epididymis?

- Swelling includes the testis. Think of testicular cancer or old clotted haematocele.
- Swelling is confined to the epididymis. Think of tuberculous and chronic non-specific epididymitis.

FRACTURES AND DISLOCATIONS GENERAL PRINCIPLES

A fracture is defined as a structural break in the normal continuity of a bone. It is usually caused by trauma.

CHAPTER CONTENTS

Aetiology

Fractures are usually traumatic, but are sometimes secondary to a disease in the bone (pathological) or due to excess fatigue of the bone (stress).

Traumatic fractures

The mechanism of injury differs according to the type of bone (tubular or cancellous).

A tubular bone may be broken either by direct or indirect violence or by muscular violence.

A. Direct injury, e.g., fracture of the tibia if the bumper of a motor car strikes the bone. The fracture occurs at the point of impact. In the leg or forearm, if both bones are involved, they are usually fractured at the same level.
B. Indirect violence, e.g., fracture of the tibia if a skier twists his body while the ski is held fixed. The bones break at a distance from the point of impact. If both bones of the leg or forearm are involved, they are usually fractured at different levels.
C. Muscle violence due to uncoordinated muscular contraction.

A cancellous bone may be fractured either by compression or by tension.

A. **Compression fractures**, e.g. if a patient falls from a height and lands on his heels. The compressive force traveling up the leg and through the trunk may produce a crush fracture of a vertebral body, of the tibial or femoral condyles, or of the os calcis.
B. **Traction injuries**: These occur only in cancellous bones to which a ligament or tendon is attached, e.g., the medial malleolus and the patella.

Pathological fractures

These are due to abnormal fragility of the bones, which break as a result of trivial violence. The causes are:

A. **Local bone diseases** as osteomyelitis, cysts, primary tumours or secondaries.
B. **Generalized bone** diseases as osteogenesis imperfecta, osteoporosis, hyper-parathyroidism, Paget's disease and multiple myelomatosis. In these diseases multiple fractures are liable to occur.

Stress fractures

Stress fractures are also named fatigue fractures because they occur secondary to repeated loads applied to the skeleton, e.g., march fractures which occur in the second and third metatarsals due to prolonged walking.

Clinical features and diagnosis

Clinical diagnosis

History

1. There is usually history of injury except in cases of stress fractures and some cases of pathological fractures.
2. Immediately after the fracture has occurred the patient suffers local pain which ranges from mild to severe.

Examination reveals one or more of the following signs:

1. **Swelling and ecchymosis** due to effusion of blood from the broken bone ends and torn soft tissues.
2. **Deformity** due to displacement of the fragments by either
   a. The causative violence
   b. The weight of the limb; or
   c. The action of muscles.
3. **Tenderness**. This is elicited over a localized point on the surface of the bone.
4. **Loss of function** of the injured part which varies from minimal to complete.
5. **Crepitus** is felt as a sensation of grating when the bone ends are moved against each other.
6. **Abnormal mobility** in the line of the bone, except in incomplete and impacted fractures. The last two physical signs should never be sought deliberately since their demonstration adds nothing to the diagnosis and merely causes pain and may result in further soft tissue damage and blood loss.
7. For a complete clinical diagnosis of a fracture, the **motor and sensory innervation** of the limb, and the **peripheral circulation** should be examined to record and treat any injury of nerves or blood vessels.

Radiological diagnosis

The minimum radiographic examination is only complete when radiographs of the whole bone have been obtained, including the joints in which it participates. X-rays should also be obtained in two planes at right angles to each other (routinely antero-posterior and lateral). In certain circumstances other views may also be required, e.g., oblique views for the elbow and the mandible.

Fracture description

The following points have to be assessed:

1. **Site of the fracture**. The fracture may be intra-articular, epiphyseal, metaphyseal or diaphyseal.
2. **Extent of the fracture**. According to their extent fractures are classified into:
   - Complete fractures in which the bone is broken into 2 or more fragments which tend to become displaced with loss of opposition and alignment.
   - Incomplete fractures in which the bone is incompletely broken so that displacement cannot occur, as in fissures, greenstick fractures (Fig. 49.1) and subperiosteal cracks of children.
3. **Fracture line** (Fig. 49.2). According to the direction of the fracture line, fractures may be:
   - Transverse usually due to direct violence.
   - Oblique or spiral due to indirect violence.
   - Comminuted due to severe compression. There are more than two bone fragments.

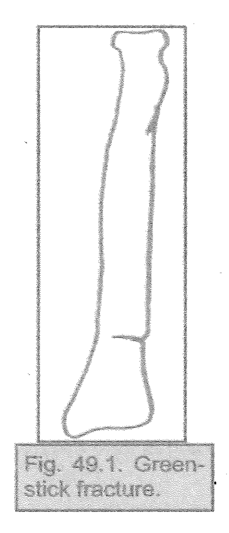


Fig. 49.1. Green-stick fracture.

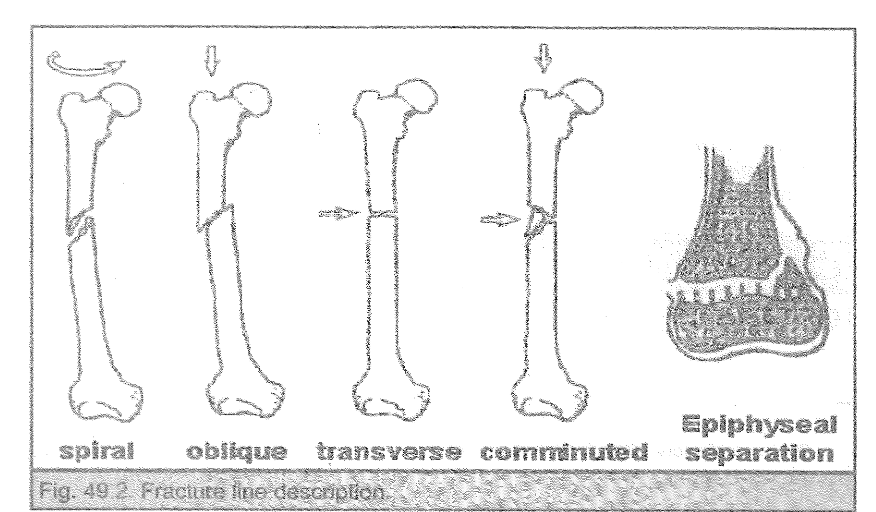


Fig. 49.2. Fracture line description.

- Avulsion due to separation of a bony process with its attached muscles.
- Epiphyseal separation in children always occurs on the metaphyseal side of the epiphyseal cartilage so that the separated epiphysis always includes the epiphyseal cartilage with a triangular fragment from the metaphysis.

4. **Displacement**. This refers to the deformity which may be present following a fracture or dislocation and describes the "position of the distal component in relation to the proximal one". The causes of displacement and deformity are the effect of the trauma, the force of gravity, the power of the surrounding muscles and the movement of the patient during transport. The following are possible types of displacement (Fig. 49.3):
   - **Lateral displacement**, the distal fragment deviates to one side with loss of opposition.
   - **Angulation**: This refers to loss of the normal longitudinal axis of the shaft. It may occur in one of four directions, anterior, posterior, medial and lateral.
   - **Over-riding**, the distal fragment overlaps the proximal fragment with shortening of the limb.
   - **Rotation**, the distal fragment is rotated along its long axis.
   - **Distraction**, the fragments are separated either by excessive muscle pull at the time of injury (rare) or more commonly by over vigorous traction during treatment.
   - **Depression**, a fragment of bone is displaced, e.g. a skull fracture. Impaction, the distal fragment is forcibly driven into the proximal fragment so that abnormal mobility and crepitus cannot be elicited.
5. **Stability**. A stable fracture is a fracture where further

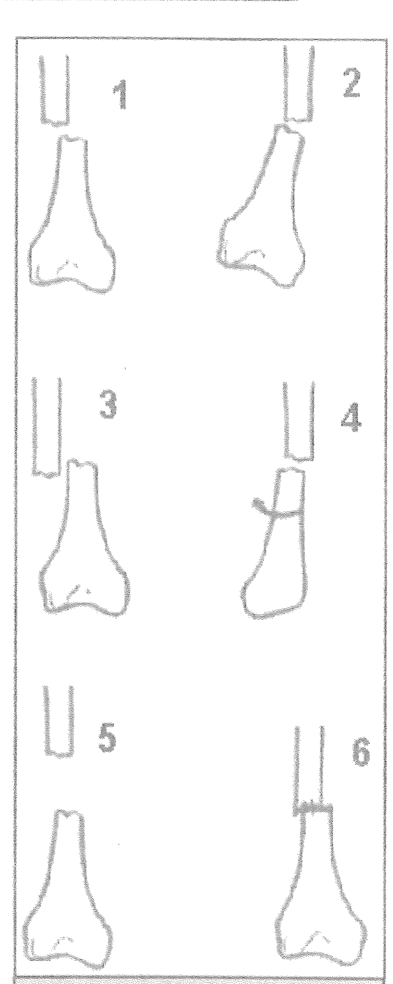


Fig. 49.3. Fracture displacement:

1. Lateral displacement.
2. Angulation.
3. Over-riding.
4. Rotation.
5. Distraction.
6. Impaction.

displacement after reduction is unlikely. The reverse is true for unstable fracture. Factors controlling stability are:

- Line of the fracture, e.g., oblique, spiral and comminuted fractures are unstable.
- Muscle pull.
- Integrity of the supporting ligaments.

6. **Skin damage**. According to the condition of the overlying skin fractures are classified into:
    - **Simple (closed) fractures**, in which the skin surface is intact.
    - **Compound (open) fractures**, in which a laceration in the skin or mucous membrane communicates with the fracture haematoma. Bacteria can therefore reach the fracture site. The skin wound may be caused by a fracture fragment penetrating the skin from its deep surface "compound from within" or by direct injury to the skin "compound from without" (Fig. 49.4). The chances of significant bacterial contamination of the fracture are greater in the latter, and hence fractures which are compound from without have a poorer prognosis. However, the distinction between the two types is only of academic interest and has no influence on the treatment. A break in the skin surface may be due to skin death from ischaemia rather than to a laceration. Fractures of subcutaneous bones with displacement may lead to pressure on the deep surface of the skin by a bony fragment and thus endanger the circulation to the skin. Alternatively, injuries causing fractures by direct violence may crush the blood vessels in the skin but this may not be obvious at the time of the first examination. During the following two to three weeks the skin may slough and if this possibility is not borne in mind, a fracture may be encased in a plaster of Paris splint in which the skin may slough unnoticed.

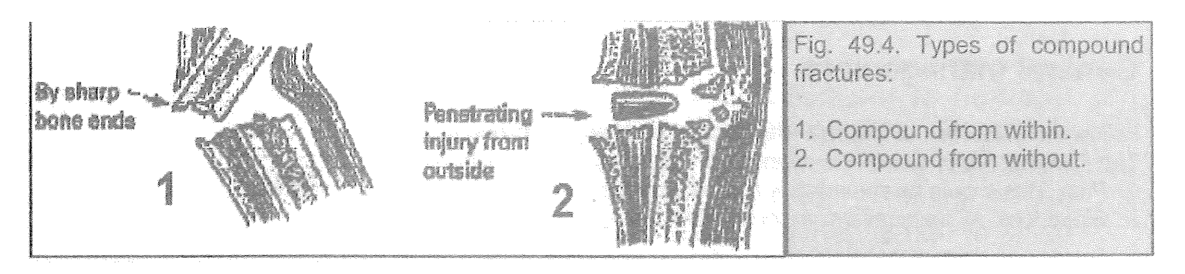


Fig. 49.4. Types of compound fractures:

1. Compound from within.
2. Compound from without.

Table (49.1): Illustrates the classification of compound fractures

Type of fracture	Skin wound	Soft tissue drainage	Infection risk
Type I	↓7cm	Minimal	↓2%
Type II	↑7cm	Moderate	10%
Type III	Any size	Severe e.g. war wounds or RTA	↑10%

Fracture healing

Phases (Fig. 49.5)

1. **Repair by granulation tissue**. A haematoma forms between the bone ends underneath the raised periosteum. Vascular granulation tissue invades the haematoma and bridges the fracture gap. This stage lasts for a few weeks.
2. **Union by primary callus.** Trabeculae of cartilage invade and replace the granulation tissue. Bone cells and matrix gradually appear leading to the formation of an irregular mass of vascular bone and calcified cartilage at the site of the fracture (primary callus). This callus first appears on the outer aspect of the fragments (external callus), then along the medullary canal (internal callus) and finally between the cortex of the bone ends (intermediate). This stage ends at 2-3 months.
3. **Formation of mature bone**. The intermediate callus is gradually replaced by lamellar trabeculae laid down along the lines of stress and strain. The external and internal calluses are absorbed with restoration of the medullary cavity. This stage ends at 4-6 months.

Fig. 49.5. Stages of fracture healing.
1. Fracture haematoma and granulation tissue
2. Start of callus formation.
3. External, internal and intermediate callus.
4. Mature bone and remodelling

Factors affecting union of fractures

1. **Age.** Children have their fractures healed in a much shorter time than adults. Elderly people have senile osteoporosis which retards the union of fractures.
2. **Type of fracture**. Long oblique and spiral fractures heal more rapidly than transverse fractures.
3. **Position of the fragments**. If the fragments are impacted, union occurs rapidly. Distraction of the fragments markedly delays healing.
4. **Vascularity of the fragments**. Certain fractures are notorious for delayed healing due to impaired blood supply. A famous example is an intracapsular fracture of the neck of femur where injury of blood supply occurs leading to ischaemia and avascular necrosis of the femoral head. Fractures of the lower thirds of the tibia and ulna are also liable to delayed union.
5. **Immobilization of the fragments.** This is vital for proper healing. Lack of proper immobilization leads to repeated movement at the fracture site interfering with the process of union.
6. **Infection.** This is disastrous to the healing process, as the granulation tissue is destroyed by infection and the bone ends are decalcified leading to non union.

Treatment of fractures

The management of a fracture can be discussed under two headings: (1) the management of the patient as whole and (2) the local management of the fracture itself.

General management

Follow the guidelines of trauma life support that are described in chapter 2.

The following are of special importance with the treatment of fractures.

1. **Pain**. This should be immediately relieved by local splinting (at the accident site) and by analgesics.

2. **Blood loss**. All fractures are associated with some blood loss that might be considerable as in fractures of the major long bones, the pelvis and the spine. This loss, however, may not be immediately obvious. Thus, a patient suffering a fracture of the pelvis or the shaft of the femur can lose 2 litres of blood into the surrounding tissues without any obvious swelling or bruising. Such blood loss must be replaced.
3. **Attention to associated injuries**, e.g. injuries of the urinary bladder in fractures of the pelvis. The possibility of such injuries must always be borne in mind since they may be missed unless they are deliberately sought at the time of the first examination.
   N.B: The management of internal haemorrhage and visceral injury takes priority over a limb fracture.
4. Tetanus toxoid and antibiotics: These should be used in compound fractures.

Local treatment

The principles of treatment of a fracture are: reduction, fixation and rehabilitation.

Reduction of the Fracture

Aim. Reduction of a fracture implies restoration of normal, or perhaps more correctly, acceptable anatomy because absolutely normal anatomy is not always achieved, especially when treating fractures by closed means.

Reduction is not necessary when the displacement is trivial (e.g. a fractured metacarpal; the fragments being splinted by neighbouring bones and soft tissue) or when the displacement is of a nature that will leave no functional or cosmetic disability (e.g. most fractures of the clavicle).

Time. Early reduction of a fracture before the part gets swollen is preferable to delayed reduction after a few days. Reduction is urgent when the fracture is complicated by vascular or nerve injury. Accuracy of reduction is desirable in any fracture, particularly fractures involving articular surfaces. Angulation, rotation and overlap are not permissible especially in the lower limb and forearm. Partial side displacement is accepted in a transverse fracture of the shaft of a long bone.

Methods of reduction

1. Closed reduction using one of the following methods:
   a. Gravity. The muscle power of the upper limb is not very strong, and fractures like surgical neck or shaft of humerus can be reduced relying on the effect of gravity by placing the limb in a collar and cuff. A plaster of Paris cast may be applied to increase the weight of the limb and prevent the side-to-side displacement.
   b. Closed manipulation. Closed reduction is better done under general anaesthesia, so that, the procedure is painless and to have adequate muscular relaxation for perfect reduction.
   c. Traction. This is particularly used for fractures of the long bones of the lower limb, e.g. fracture shaft femur. Skin or skeletal traction is used to keep reduction of the fracture.
2. **Open reduction:** Open (surgical) reduction is resorted to in the following situations:
   a. Inability to achieve closed reduction due to interposition of soft tissues between the fractured segments.
   b. If it is impossible to reduce the fracture because of inability to hold one of the fracture fragments, e.g. fracture of the anatomical neck of the humerous with anterior dislocation of the head of the humerous which is inaccessible.
   c. Intra-articular fractures as closed reduction will be inaccurate.

d. Late unreduced fracture.
e. When internal fixation is needed.

Stabilization (fixation) of the fracture

After reduction of the fracture, the next step is to keep the two ends of the fracture in this position until solid healing occurs. Methods available for splinting include:

1. **External fixation**
   a. Plaster of Paris. The plaster should include the joints adjacent to the fracture site. The advantages of this method are that it is cheap, safe and does not need extensive facilities. However, Plaster-of-Paris cast has some disadvantages:
      i. Joint stiffness.
      ii. Muscular atrophy as the muscles underneath the plaster remain immobilized for a long time. Inability to maintain perfect reduction during the immobilization period.
      iii. Liability to cause compartment syndrome or Volkmann's ischaemic contracture. In the first few hours after application of the Plaster, edema occurs at the fracture site and as the plaster is inelastic, the local pressure will increase. Venous obstruction occurs first and if the condition is not treated is followed by arterial obstruction. The condition will end in ischaemic necrosis of the muscles (Chapter 16). To avoid this serious complication, the limb should be well padded with wool and the plaster should not completely surrounded the limb. The patient should be kept in the hospital for 24 hours to observe the circulation in the periphery of the limb. Should any sign of ischaemia appear, the plaster should be immediately removed.
   b. **Traction**. The fracture may be immobilized by applying traction on the involved limb. Traction may be either skin or skeletal traction (Fig. 49.6). In skin traction, an adhesive bandage is applied to the skin and then a weight is affached to the bandage. The disadvantage of this method is that only a small weight not more than 2.25 Kg can be applied and so it is suitable only for children. In skeletal traction, a special pin is passed into the bone and then a weight is applied to the pin. This method is used in adults for fractures of the lower limb. Immobilization should continue until there is evidence of clinical healing of the fracture.
   c. External skeletal fixators. This method is useful in compound fractures. Special pins are introduced in the bone proximal and distal to the site of the fracture and the pins are connected to a special metal frame (Fig. 49.7). The procedure avoids operative internal fixation with the possibility of infection and it leaves the wound for inspection. The procedure is very useful when the skin condition does not allow internal fixation.

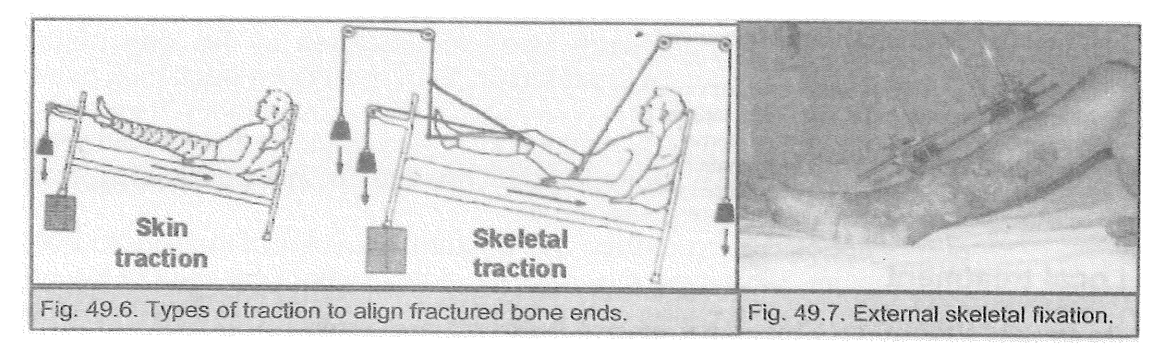


Fig. 49.6. Types of traction to align fractured bone ends.

Fig. 49.7. External skeletal fixation.

2. **Internal fixation**

Methods (Fig. 49:8)

a. Wires.
b. Screws
c. Plate and screws.
d. Intramedullary nail.
e. Compression screw and plate for a fractured neck of femur.

Indications

1. Difficult fractures
    i. Those prone to non-union, especially the femoral neck.
    ii. Those prone to malunion, e.g. fracture-sublaxation of the ankle and wrist which are often unstable and mid-shaft fractures of the forearm.
    iii. Those prone to be pulled apart by muscle action, e.g. transverse fractures of patella and olecranon.
2. Pathological fractures. Fractures due to metastatic deposits or in osteoporotic bones are best treated by internal fixation to accelerate healing
3. Multiple fractures. With two major fractures in one limb, fixation of one facilitates closed treatment of the other.
4. Unstable fractures.
5. Nursing difficulties
    i. Elderly patient.
    ii. Patients with traumatic paraplegia.
6. Associated soft tissue injuries, e.g. vessels or nerves. In this situation if facilities are available vascular or nerve repair is done and the fracture is internally fixed. Fracture fixation should precede vascular or neural repair, or else the site of repair would be subject to disruption.

Advantages

1. Very accurate reduction of the fracture segments.
2. Strict immobilization of the fractured bones.
3. Early mobilization of the patient thus avoiding all the problems of prolonged immobilization particularly in elderly persons.
4. Protection of vascular anastomoses in patients with vascular injury.

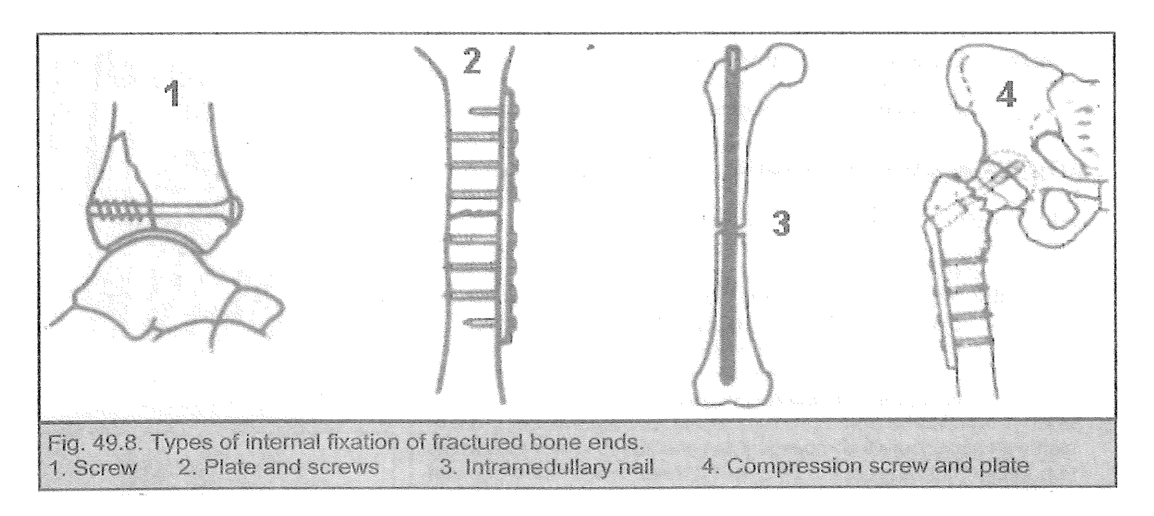


Fig. 49.8. Types of internal fixation of fractured bone ends.
1. Screw 2. Plate and screws 3. Intramedullary nail 4. Compression screw and plate

Rehabilitation

The aim is restoration of function of the injured part, and the patient as a whole. All unsplinted joints must be extensively used from the very start. The muscles controlling the

joints are kept active by exercises. After removal of the splints, residual joint stiffness is treated by intensive but graduated exercises.

Management of compound fractures

Treatment is surgical. The aim is to convert the compound fracture into a simple one. If infection occurs in a compound fracture, it will have very serious consequences. The primary aim of the operation is to clean the wound and to obtain skin closure as rapidly as possible:

- Treatment is done in an operating theatre.
- General anaesthesia.
- The wound is thoroughly cleansed.
- The deep fascia is widely opened.
- Any foreign bodies, necrotic tissue, necrotic muscles, or necrotic skin edges are removed.
- Any soft tissue injury is dealt with according to the rules (chapter 1).
- Bleeding points are tied but an injury to a major vessel needs to be repaired.
- The skin may or may not be closed according to circumstances (Chapter 1). As regards the fracture itself some form of external stabilization by a plaster slab or external skeletal fixators is used. All forms of internal fixation should be avoided for fear of inviting infection.
- Prophylactic antibiotics and prophylaxis against tetanous follow the general principles.

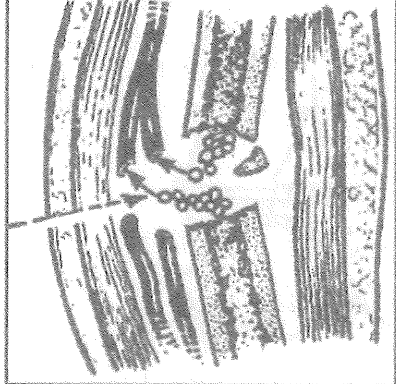


Fig. 49.9. Fat embolism.

Complications of fractures

These are classified into general or local. The latter are further divided into early and delayed local complications.

General complications

1. **Shock**. Neurogenic or hypovlaemic shock may accompany major fractures as fracture spine, pelvis or femur. The blood loss in a fracture pelvis may amount up to 2-2.5 litres.
2. **Fat embolism** (Fig. 49.9): This condition may follow multiple or major fractures due to fat emboli from bone narrow or adipose tissue. It usually occurs after one day from the time of injury. Two forms exist:

    a. Cerebral: the patient is drowsy, restless and subsequently may be comatose.

    b. Pulmonary: there is cyanosis, frothy sputum or heart failure.

    Investigations: Examination of the sputum and urine for fat droplets.

    Treatment: O_2 mask, heparinization and low molecular weight dextrans.

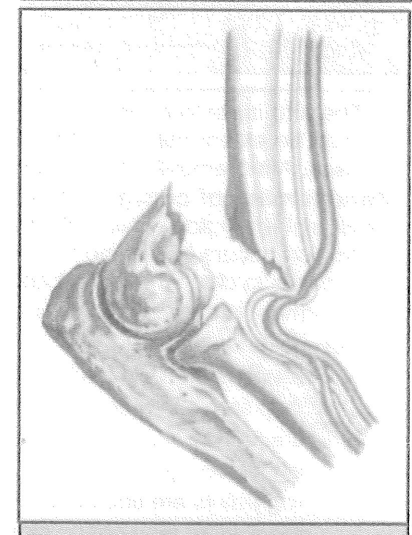


Fig. 49.10. Brachial artery injury caused by a supracondylar fracture of the humerus.

3. **Respiratory complications**. Prolonged recumbency particularly in elderly persons may lead to respiratory complications including aspiration pneumonitis or pulmonary

embolism. Early mobilization of patients following recent techniques of open reduction and internal fixations avoids these problems.

4. Deep vein thrombosis.
5. **Urinary calculi.** Prolonged immobilization leads to demineralization of the skeleton with the formation of calcium phosphate calculi. High fluid intake and early mobilization are recommended.
6. Bed sores may develop secondary to prolonged immobilization especially in elderly persons. Frequent change of the position of the patient in bed, massage, assurance of dry bed sheets and the use of an air-mattress are essential.
7. **Tetanus**. The tetanus organism flourishes only in dead tissues. It produces an exotoxin which passes to the central nervous system via the blood from the infected region, e.g. in compound fractures.

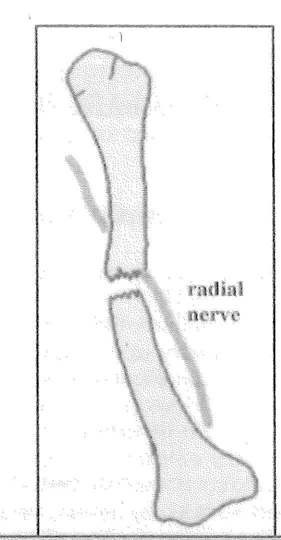


Fig. 49.11. Radial nerve injury by a fractured shaft of humerus.

Early local complications

1. **Skin injury**. The fractured bone ends may injure the skin from inside (internal compound fracture), but fortunately the risk of infection in these cases is not high.
2. **Vascular injuries** (Fig. 49.10). The fractured bone ends may injure the adjacent vessels, e.g. injury of the brachial vessels following a supracondylar fracture of the humerus (see Volkmann's ischaemic contracture in chapter 16) or injury of the popliteal vessels as a consequence to supracondylar fracture of the femur. The artery may also be damaged by a tense haematoma or by contusion with secondary thrombosis. It is essential to check the circulation in the distal part of the limb and if there is suspicion of a vascular injury, an angiography may be performed. If an arterial injury is diagnosed, open reduction and internal fixation are performed prior to the vascular repair. Venous injuries are also repaired.
3. **Nerve injuries**. Any adjacent nerve may be injured either by excessive traction or compression leading to neurapraxia, axonotemesis or less commonly by being torn leading to neurotemesis. It is essential in every patient with a fracture to check for the possible occurrence of a nerve injury. Examples include circumflex nerve injury in fracture neck humerus or radial nerve injury in fracture shaft humerus (Fig. 49.11).
4. **Tendon or muscle injury**.
5. **Infection**. This serious complication may complicate compound fractures. It leads to delayed healing, non union or osteomyelitis.
6. **Avascular necrosis of bone**. Certain fractures may

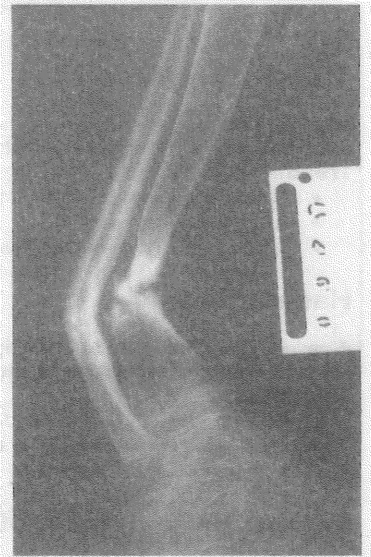




Fig. 49.12. Two examples of malunion.
Upper. Both bones of forearm.
Lower. Pelvis. The resulting deformity may interfere with vaginal delivery

damage the blood supply to some areas of bones leading to avascular necrosis. The commonest example is intracapsular fracture of the neck of the femur which may lead to avascular necrosis of the head of the femur. Consequences of this necrosis are delayed union or non-union of the fracture or late development of osteoarthrosis of the hip joint.

7. **Visceral injury**. A fracture of the pelvis may be complicated by injury to the bladder or urethra.

Delayed local complications

1. **Malunion** (Fig. 49.12). This means union but with some deformity, e.g. angulation. It is due to lack of perfeat reduction or failure of stabilization. Open reduction and internal fixation avoid this complication. Malunion may lead to cosmetic or functional disability.
2. **Delayed union and non-union** (Fig. 49.13) are due to:
   a. Impaired blood supply of one or both fragments, e.g. in intra capsular fracture neck femur. This will lead to poor invasion of the fracture haematoma by cells with osteogenic potential. The low oxygen tension due to poor vascularity interferes with the ability of these cells to produce bone.
   b. Inadequate immobilization which leads to repeated shearing strain on the granulation tissue.
   c. Interposition of soft tissue between the fragments.
   d. Overdistraction of the fragments due to excessive skeletal traction.
   e. Infection which destroys the granulation tissue.
   f. Severe initial trauma with compound fracture and extensive damage of the soft tissues. This allows diffusion of the fracture haematoma into the soft tissues. Moreover, extensive soft tissue damage produces impairment of the blood supply of the bone fragments.
   g. Pathological fractures, e.g. due to malignant disease.

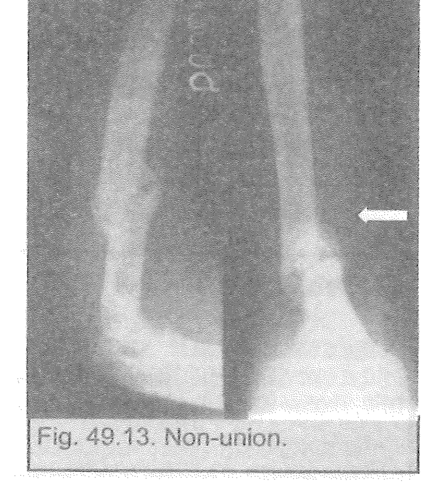


Fig. 49.13. Non-union.

   In delayed healing the bone ends are decalcified and the fracture line is widened into a gap full of fibrous tissue. Clinically there is still abnormal movement and tenderness at the site of the fracture. There is still a chance of union with prolonged, uninterrupted immobilization.
   In non union the bone ends are sclerosed and the medullary canal is closed by a dense bone. Spontaneous healing is impossible. The treatment of delayed union and non-union should be by some form of internal fixation and application of autogenous bone graft obtained from the cancellous tissue of the iliac rest.
3. **Sudek's atrophy**. This is a syndrome of unknown aetiology in which there is osteoporosis, swelling of the soft tissue, vascular stasis, pain and joint stiffness complicating a fracture. It is supposed to be due to reflex vascular stasis or due to reluctance of the patient to use the limb after removal of the splint. It is most often seen after Colles' fracture, Treatment includes splinting, sympathetic block, analgesics, and physiotherapy.

4. **Myossitis ossificans**. This is due to extensive stripping of the periosteum and ossification of the subperiosteal haematoma or hetrotropic bone formation in adjacent muscles. It is most often seen after dislocation of the elbow, shoulder or hip. Treatment is by immobilization to allow the haematoma to settle down and the newly formed bone to resorb. Massage should be avoided.
5. **Joint stiffness and osteoarthrosis** may occur after prolonged immobilization and after intra-articular fractures. Certain joints as the elbow, shoulder and hip are particularly prone to this problem. Damage to the articular cartilage or avascular necrosis of the bone following an intra-articular fracture may lead to osteoarthrosis.
6. **Growth disturbance** may occur it the fracture affects the epiphyseal growth plate in children.
7. **Osteoporosis** due to prolonged immobilization.

Injuries of Joints

Ligament injuries

The ligaments supporting a joint may be injured. The injury ranges from a sprain in which only some of the fibers of the ligament are injured to a complete tear of the ligament. In sprain there is limited movement, severe pain and tenderness at the site of injury, but the joint is stable. In complete tear of the ligament there is haemarthrosis and instability of the joint. Examination under anaesthesia may be needed to assess the stability of the joint.

Treatment

- Sprain. The joint is firmly strapped until the pain subsides.
- Complete tear
  - Haemarthrosis is aspirated.
  - Plaster of paris cast for 6-8 weeks.
  - Later active exercises avoiding tension on the ligament.

  In young individuals it is better to do surgical repair of the ligament.

Traumatic effusion

The injury stimulates the synovial membrane to produce excessive effusion. It usually occurs several hours after the injury and commonly affects the knee joint.

Treatment

- A handage is applied around the joint to limit the effusion and alleviate the pain.
- A splint may be needed.
- A tense effusion needs aspiration.

Haemarthrosis

A severe injury may lead to bleeding inside the joint. Usually there is gross ligament injury or intra-articular fracture. Shortly after the trauma, the joint is swollen with severe pain and limited mobility.

Treatment

- Aspiration of blood, and bandage of the joint.
- The joint is immobilized by a splint.
- Active muscular exercises.

Internal derangement of a joint

This refers to the various injuries which impair movements and stability of the joint. It particularly affects the knee joint where it is secondary to:

1. Injury of the medial, lateral or cruciate ligaments.
2. Injury of the menisci.
3. Loose bodies.
4. Recurrent dislocation of the patella.

Dislocation and subluxation

In dislocation the joint surfaces are completely displaced and are no longer in contact, while in subluxation there is partial disruption of the joint and the articular surfaces are still opposed.

FRACTTUES AND DISLOCATIONS OF UPPER LIMBS

In this chapter only the common injuries will be discussed.

CHAPTER CONTENTS

Fractures of the clavicle

Fracture of the middle third (shaft) (80%)

This is the commonest fracture in the whole body.

Trauma and morbid anatomy

- Clavicular shaft fracture is usually due to a fall on the outstretched hand, and less commonly due to direct blow or to a fall on the point of the shoulder.
- The fracture almost always occurs in the middle third, because it is the thinnest part of the bone which is further weakened by the junction of the two main curves of the shaft.
- The potential deforming forces are the weight of the arm that leads to downwards and inwards displacement of the outer fragment and the pull of the sternomastoid muscle that leads to upwards displacement of the proximal fragment (Fig. 55.1).

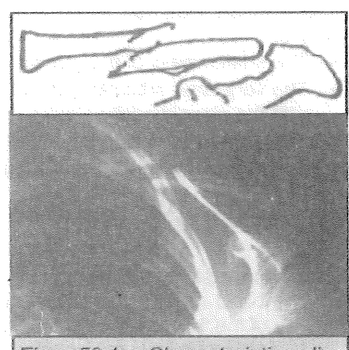


Fig. 50.1. Characteristic displacement in fracture of the middle third of the clavicle.

- In children the fracture is often of the greenstick variety.

Complications

1. Malunion is common but is of no functional significance and is rarely a significant cosmetic problem.
2. Non-union is uncommon.
3. Injury of the subcalvian vessels and brachial plexus.

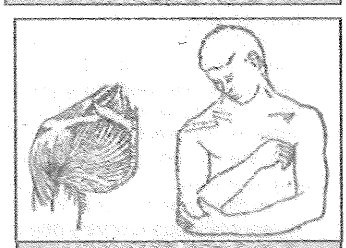


Fig. 50.2. Characteristic posture in fracture of the middle third of the clavicle.

Clinical features

The shoulder is dropped and deformed, and the patient usually supports the elbow with the opposite hand and bends his head to the affected side to relax the sternomastoid muscle (Fig. 50.1).

Treatment

- A broad arm sling (Fig. 50.3) for 3 weeks and nalgesics.
- Internal fixation is rarely needed when there is associated vascular or nerve injury.

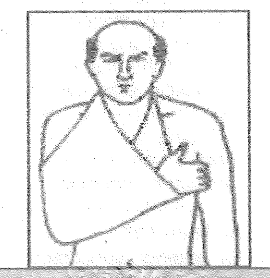


Fig. 50.3. Broad arm sling for clavicular fractures.

Dislocation of the shoulder joint

The shoulder joint has sacrificed stability for mobility. Its instability is due to:

- The shallowness of the glenoid cavity.
- The very large articular surface of the humeral head in comparison with the glenoid cavity.
- The laxity of the capsule.
- The lack of support by strong ligaments and muscles.

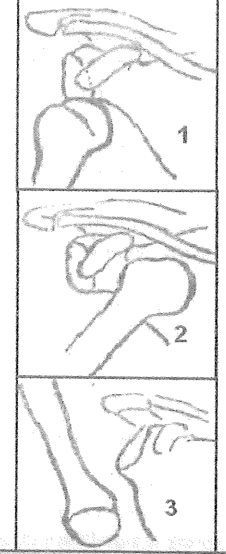


Fig. 50.5. Types of shoulder dislocation.
1. Subcoracoid.
2. Subclavicular.
3. Luxatio erecta.

Anterior dislocation

Trauma and morbid anatomy

The head of the humerus usually dislocates forwards to assume one of the following positions (Fig. 50.5):

1. Subcoracoid (commonest).
2. Subclavicular.
3. Luxatio erecta is an anterior dislocation in which the arm is abducted above the head.

The injury is produced by forced extension and external rotation of the abducted arm. The head of the humerus is forced against the capsule of the shoulder and either ruptures its anterior aspect or avulses the labrum glenoidal from the glenoid. The humerus passes over the anterior margin of the glenoid and in so doing the posterior part of the articular surface of the head may suffer a compression fracture. Once in its new subcoracoid position, the humerus is locked in this position by muscle spasm.



Complications

1. Axillary nerve injury. In most cases it is a neurapraxia and spontaneous recovery often occurs.
2. Avulsion of the supraspinatus tendon is discovered by inability of the patient to initiate abduction. It needs repair of the musculotendinous cuff.
3. Associated greater tuberosity or humeral neck fracture.
4. **Recurrence** is a common problem. It is due to detachment of the labrum glenoidal and the anterior capsule from the anterior margin of the glenoid. A pouch is formed in front of the bare neck of the scapula into which the humeral head slips easily. Clinically redislocation occurs with increasing ease and frequency following relatively minor everyday actions such as combing the hair. Reduction is equally easy and usually the patient can reduce the dislocation himself. When the arm is abducted, externally rotated and gently moved backwards, the patient feels that the dislocation is imminent.



Fig. 50.6. Anterior shoulder dislocation.

Clinical features

- In anterior subcoracoid dislocation the outer aspect of the shoulder is flattened and the head is felt displaced in the subcoracoid region. The arm appears to take origin from a point under the junction of the middle and outer thirds of the clavicle.
- The shoulder cannot be moved and the arm is held in a position of slight abduction.
- The patient often appears supporting the elbow of the injured arm by the other hand.

- The distance between the point of the elbow and the axillary skin is reduced, and the axillary concavity is obliterated.
- X-rays in anteroposterior views are diagnostic (Fig. 50.6).

Treatment

Reduction and immobilization

- Reduction. Under general anaesthesia with muscle relaxation closed reduction of the dislocated head is done using the Kochers technique (Fig. 50.7).
    - o Downward traction on the arm to disengage the head.
    - o External rotation of the humerus to overcome spasm of the subscapularis muscle.
    - o The upper third of the humerus is levered outwards by adducting the humerus bringing the elbow across the chest.
    - o The shoulder is then internally rotated carrying the hand to the opposite shoulder and the head to its original position.

  Reduction is confirmed by putting the shoulder through a full range of movement under anaesthesia.

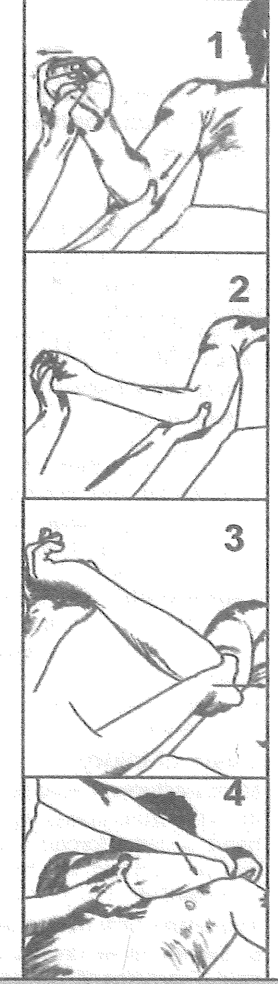


Fig. 50.7. Kocher's method for reduction of anterior shoulder dislocation.

- The arm is then immobilized in a sling and bandaged to the side so as to maintain full adduction and internal rotation of the shoulder for 3 weeks to allow healing of the torn capsule and to minimize the tendency for recurrence. At the end of 3 weeks, the sling is removed and for three more weeks progressive mobilization of the shoulder is done avoiding only external rotation in abduction (the position in which the shoulder dislocates).

Treatment of recurrent shoulder dislocation

Operation is indicated when the disability is great.

1. Putti-Platt operation. The idea is to limit external rotation of the shoulder by capsulorraphy and shortening of the subscapsularis muscle by overlapping.
2. Bankart operation. This has better results but is technically more demanding. The original lesion in the capsule or glenoidal labrum is displayed and is repaired by reattaching the labrum by suturing it to the bony rim of the glenoid.

Posterior dislocation

This is not common. The head slides backwards to lie below the acromion (subacromial dislocation) or in the infraspinous fossa of the scapula (subspinous dislocation). It is usually caused by forced internal rotation of the abducted arm or by a direct blow on the front of the shoulder. It should always be suspected after an epileptic fit or an electric shock.

Fractures of the humerus

Fractures of the proximal humerus

Type of patient

Fractures of the proximal humerus are common in the elderly and are usually associated with osteoporosis. They may result from minor trauma. In younger adults they require a considerable force and fracture dislocations may occur. In children, before skeletal maturity, proximal humeral fractures may involve separation of the proximal humeral epiphysis.

Classification

The prognosis of these fractures depends upon the degree of displacement and the number of fracture fragments. These parameters are best shown by the Neer's 4 segments classification of proximal humeral fractures.

Fractures of the proximal humerus can occur between all four of its major segments:

1. Articular segment or fractures occurring through the anatomical neck.
2. Greater tuberosity.
3. Lesser tuberosity.
4. Surgical neck.

The classification then identifies the fractures as one-part, two-part, three-part and four-part fractures, depending upon the number of segments displaced. More than one centimeter of separation or 45 degrees of angulation of any segment is considered a displaced fragment.

One-part fracture: no displacement (even though a fracture line may exist between any number of segments).

Two-part fracture one segment is displaced.

Three-part fracture two segments are displaced.

Four-part fracture three segments are displaced (Fig. 50.9).

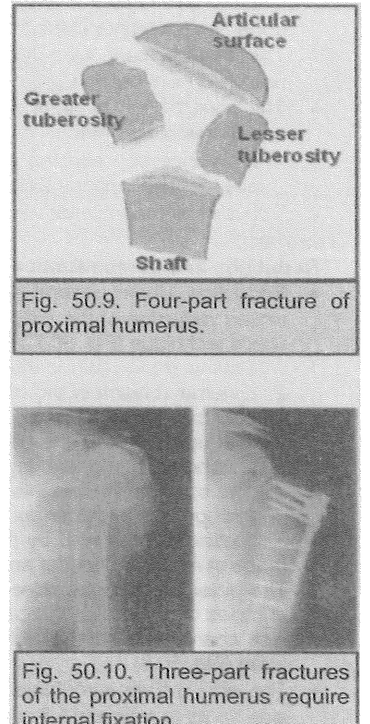


Fig. 50.9. Four-part fracture of proximal humerus.

Fig. 50.10. Three-part fractures of the proximal humerus require internal fixation.

Mechanism of injury

- Most fractures, particularly those involving the surgical neck of the humerus, occur as a result of a fall on the outstretched hand causing forced abduction, extension, and external rotation and driving the humerus upwards against the acromion and glenoid.
- The anatomical neck may fracture following a fall onto the point of the shoulder driving the humerus directly against the glenoid. Fractures of the greater tuberosity also usually result from falls on the shoulder.

Clinical features

- The patient complains of pain in the shoulder and inability to move the joint.
- In minor impacted fractures of the surgical neck, limited movement may be possible.
- The diagnosis is made radiologically.

Treatment

One-part fractures

Approximately 80 percent of fractures of the proximal humerus are undisplaced because the fragments are stabilized by the intact rotator cuff and the periosteum. These

fractures are simply treated by external immobilization using a sling until pain has subsided sufficiently to allow early active mobilization of the shoulder to be commenced in order to prevent shoulder stiffness.

Two-part fractures

- Fractures of the greater tuberosity. Open reduction and internal fixation, using screw or wire, followed by arm rest in a sling. Arm abduction is not allowed for at least three
  weeks postoperatively.
- Fractures of the lesser tuberosity usually require no treatment other than immobilization.
- Fractures of the surgical neck. These can usually be treated by closed manipulation or simply by application of a collar and cuff to allow gravity to reduce the fragments. This should be followed by early passive mobilization to prevent shoulder stiffness.

Three-part fractures

- Open reduction and internal fixation (Fig. 50.10) along with repair of the rotator cuff.

Four-part fractures

- These have a high incidence of avascular necrosis of the humeral head and are probably best treated by prosthetic replacement of the head (Fig. 50.11), cuff repair and wire loop fixation of the tuberosities.

Fig. 50.11. Four-part fractures of the proximal humerus require prosthetic replacement of the head.

Complications

1. Shoulder stiffness. This is common especially in the elderly who may never regain a full range of movements. Treatment should, therefore, be directed towards early passive followed by active mobilization.
2. Axillary nerve injury (Fig. 50.12).
3. Malunion is not uncommon but compatible with excellent functions in many cases.
4. Avascular necrosis of the humeral head.
5. Nonunion (not common).
6. Dislocation of the shoulder: In these cases the dislocation should be treated first.

Fig. 50.12. Circumflex nerve injury causes a patch of hyposthesia over the lower half of the deltoid.

Fracture of the proximal humeral epiphysis in children

- In children even a complete fracture separation of the epiphysis with gross displacement can be managed non-operatively (collar and cuff for three weeks) because extensive remodelling occurs thus correcting any deformity.
- In adolescents the potential for remodelling is limited; therefore the fracture should be reduced. An attempt at closed reduction is made. If reduction cannot be maintained with the arm at the side, percutaneous pinning is indicated.

Fractures of the shaft of the humerus

Mechanism of Injury

1. Indirect trauma

a. Twisting injury to the arm producing a spiral fracture with or without a butterfly fragment.
b. Fall onto the outstretched hand.

2. Direct trauma. Direct blow over the shaft producing a transverse fracture, sometimes with a butterfly fragment.



Fig. 50.13. Fracture displacement after humeral shaft fracture.
1. Just below surgical neck.
2. Middle third.
3. Below deltoid tuberosity.

Displacement

This depends on the level of the fracture (Fig. 50.13).

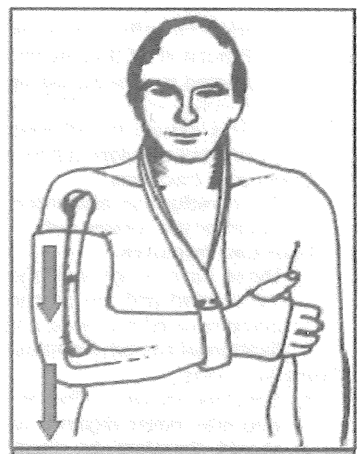
- Just below the surgical neck. The proximal fragment is abducted by the supraspinatus and the distal fragment is adducted by the pectoralis major.
- Middle third. The upper fragment is adducted and pulled inwards by the pectoralis major and the lower is abducted by the deltoid.
- Below the deltoid tuberosity. The proximal fragment is abducted by the deltoid and the distal fragment is adducted and pulled upwards by the coracobrachialis.

Complications

1. Radial nerve injury (Fig. 49.11). The treatment is expectant because the lesion is usually in continuity and there is full recovery in most cases. It is important during treatment to prevent the development of fixed flexion of the wrist and fingers by a combination of a removal cock-up splint and passive mobilization (Chapter 18).
2. Delayed union.
3. Non union.
4. Joint stiffness.

Treatment

- Closed methods. Acceptable reduction of these fractures can usually be obtained by gravity acting on the arm and exerting steady traction which tends to correct any angulation and overriding that may occur. Most of these fractures unite easily using these closed methods in approximately 8 weeks.
  - The arm is bandaged to a U-shaped plaster slab running from the top of the acromion, down the lateral side of the arm, under the elbow and up the medial side of the arm to the axilla. For further stability the arm may, then, be bandaged to the chest wall.
  - Alternatively, a hanging cast (a complete cast extending from the axilla to the wrist) can be used. Because it is heavier, the action of gravity becomes more effective (Fig. 50.14). Its disadvantage is that the elbow cannot be mobilized during treatment.


Fig. 50.14. A hanging cast is heavy and pulls the distal fragment down to be in line with the proximal one.

- Open methods are seldom needed when it is not possible to obtain a satisfactory

reduction by the closed methods. One should then proceed to open reduction and internal fixation using a plate, or if the fracture is transverse and near the middle of the shaft, an intramedullary nail.

Fractures of the distal humerus

1. Supracondylar.
2. Condyle frctures; lateral and medial.
3. Articular surface; capitulum and trochlea.
4. Epicondyle; lateral and medial.

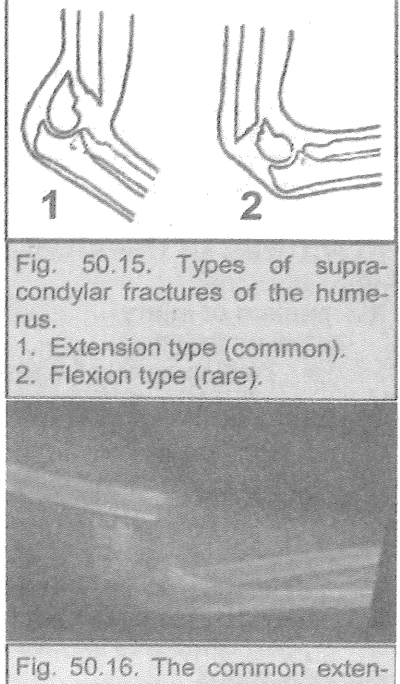


Fig. 50.15. Types of supracondylar fractures of the humerus.
1. Extension type (common).
2. Flexion type (rare).

Fig. 50.16. The common extension type of supracondylar fractures of the hymerus.

Supracondylar fractures

Supracondylar fractures are more frequent in children.

Mechanism of trauma and types

This fracture is most commonly caused by a fall onto the outstretched hand with the elbow slightly flexed to produce the so called extension type (99%), where the distal fragment is displaced posteriorly in relation to the proximal fragment. A flexion type (1%) occurs as a result of a fall on a flexed elbow (Fig. 50.15 and Fig. 50.16).

Morbid anatomy

The fracture line runs transversely through the distal metaphysis of the humerus. The fracture is greenstick in 50% and complete in 50%, of cases.

In the extension type, the displacement of the distal fragment may be minimal, but more commonly there is an obvious displacement consisting of the following elements.

1. Backward shift and angulation of the distal fragment.
2. Pronation of the distal fragment because the hand is usually pronated at the time of the trauma.
3. Internal rotation of the distal fragment as pronation produces internal rotation. As a result, the medial cortex of the distal fragment moves posteriorly relative to the medial cortex of the shaft of the humerus. On the lateral side of the fracture, however, the fragments often remain hitched to each other.
4. Medial or lateral shift of the distal fragment may occur.

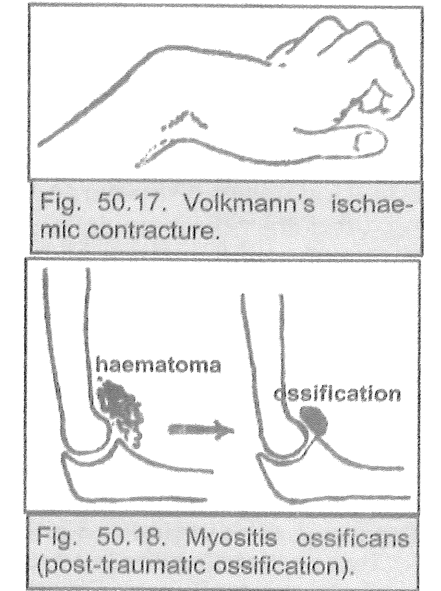


Fig. 50.17. Volkmann's ischaemic contracture.

Fig. 50.18. Myositis ossificans (post-traumatic ossification).

Complications

1. Nerve injury. Injury of the median, ulnar and radial nerves may occur. It is usually of the neurapraxia type. Conservative treatment is satisfactory in most of the cases.

2. Brachial artery injury (Fig. 49.10). This may lead to gangrene or to Volkmann's ischaemic contracture (Fig. 50.17 & Chapter 16). Fasciotomy is used to relieve an acute compartment syndrome which may occur in the presence of an adequate distal pulse.
3. Elbow stiffness.
4. Malunion may result from failure to reduce the fracture properly, producing cabitus varus, internal rotation and exten sion. The latter two will correct by remodelling, but varus angulation will not.
5. Myositis ossificans (Fig. 50.18).

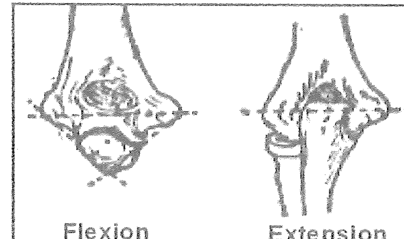


Fig. 50.19. The relationship between the two epicondyles and the tip of olecranon.
- Normally the three points are in line at extension. At flexion they form an equidistant triangle.
- With supracondylar fractures this relationship is maintained.
- With elbow dislocation this relationship is disturbed.

Clinical features

1. There is pain and swelling of the elbow region. Swelling occurs rapidly so that it may obscure the other physical signs within 3 to 4 hours.
2. The child is not able to move the elbow.
3. If significant swelling has not occurred, the posterior prominence of the point of the elbow is obvious and it is possible to palpate the medial and lateral epicondyles and the point of the olecranon so as to establish that they are normally related to each other. This differentiates supracondylar fracture from posterior dislocation of the elbow joint.
4. The vascularity of the hand on the injured side should be examined initially and then at intervals for the next 2 or 3 days. The brachial artery may be injured at the time of the trauma by the anterior aspect of the distal end of the proximal fragment. In this case the radial pulse is not felt from the start. More commonly the radial pulse is palpable at the time of the injury but is lost by swelling and flexion of the elbow following reduction.

Treatment

1. Undisplaced fractures and Greenstick fractures with angulation less than 20° require no manipulations. Fixation is achieved by using a posterior plaster slab and collar and cuff with the elbow flexed for 3 weeks.
2. Greenstick fractures with angulation more than 20° require reduction by flexion only then fixation as above.
3. Displaced fractures.
   a. Closed reduction and fixation by a posterior slab. Closed manipulation to correct the deformity should be attempted urgently under anaesthesia, by an experienced surgeon, before gross swelling has made palpation of the normal anatomy impossible. The usual extension type is fixed in flexion. Throughout the reduction procedure the radial pulse is felt and if it is obliterated by flexion, the elbow is gradually extended until the pulse retums. When the maximum degree of flexion compatible with the presence of a radial pulse has been obtained, a padded above the elbow posterior slab is applied. The reduction is checked radiographically and if satisfactory a collar and cuff is added. Some orthopaedic surgeons prefer to fix these fractures with the elbow in extension as it is said that this position allows better correction of the carrying angle and reduces the risk of arterial impairment.
   b. Closed reduction and percutaneous pinning.

c. Open reduction and internal fixation (using wires) are indicated when closed methods fail to obtain satisfactory reduction.

4. After care:
   a. The child should be admitted to hospital for observation of the circulation to the hand (usually for 48 hours). If at any time the circulation is felt to be impaired, the slab is removed and the elbow is extended. If there is no improvement, the brachial artery should be explored.
   b. The fracture unites in 3 or 4 weeks. The sling is then removed and the child's arm is gently mobilized.

There is a lot of similarity between fractures of the olecranon and those of the patella.

Dislocation of the elbow

Trauma and morbid anatomy

This usually occurs in a posterior direction, following a fall on the outstretched hand with the elbow slightly flexed. The coronoid process of the ulna passes posteriorly below the distal end of the humerus. Spasm of the triceps muscle then locks the elbow in a position of posterior dislocation (Fig. 50.21).

The forearm is usually also displaced laterally so that the commonest (80%) type of elbow dislocation is posterolateral. The dislocation can occur in other directions, anterior, medial or lateral. On occasions, the radius or ulna dislocate in opposite directions (divergent dislocation).

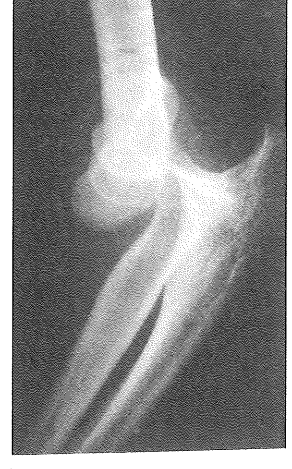


Fig. (50.21): Dislocation of the elbow

Complications

1. Associated fracture of the radial head or coronoid process.
2. Irreducible dislocations are rare and are treated by open reduction.
3. Median or ulnar nerve damage.
4. Brachial artery injury is unusual.
5. Myositis ossificans.

Clinical features

The patient is unable to move the elbow from a position of slight flex- ion. The point of the olecranon can be felt to be abnormally posterior to the humeral condyles and the triangle formed by the tip of the olecranon and the condyles is no longer equilateral. Differential diagnosis is from supracondylar fracture of the humerus.

Treatment

Under general anaesthesia traction is applied in the long axis of the slightly flexed ulna. The reduction is stable because of the bony configuration of the elbow. However, the joint should be immobilized for 3 weeks in an above the elbow posterior slab to allow for healing of the capsule and ligaments. After that the joint should be gradually mobilized.

Fractures of the forearm

These are classified as follows:

1. Fractures of the proximal ulna; olecranon and coronoid.
2. Fractures of the radial head.
3. Fractures of the shafts of radius and ulna.
4. Monteggia and Galeazzi fracture-dislocations.

Fractures of proximal ulna

Fractures of the olecranon process

Trauma and morbid anatomy

- Olecranon process fracture, is usually caused by a fall onto the point of the elbow. The olecranon will be broken by the distal end of the humerus. The fracture line is transverse and usually runs through the narrowest point of the bone. Comminution of the proximal segment may occur.
- Separation of the fracture ends depends on the integrity of the triceps expansion (Fig. 50.22).
    - If the triceps expansion is intact there will be no separation.
    - If the expansion is torn the proximal fragment will be pulled up producing a wide gap.

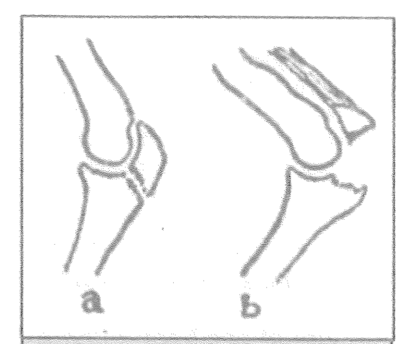


Fig. 50.22. Separation of fracture ends.
a. Intact triceps expansion.
b. Torn triceps expansion.

Clinical features and treatment

- Intact triceps expansion. There will be bruising, swelling and tenderness, but active extensive of the elbow will be possible. No gap is detected at the fracture site. The fracture does not need reduction and is treated by an above elbow plaster cast for 6 weeks followed by gentle mobilization.
- Torn triceps expansion. Here active extension is lost and the proximal segment is drawn up into the arm by the triceps muscle creating a palpable gap at the fracture site. The fracture should be treated by open reduction and internal fixation (Fig. 50.23) followed by early movement of the elbow.
- Comminuted fractures and those with, a very small proximal segment are treated by excision of the fragments and reattachment of the triceps.

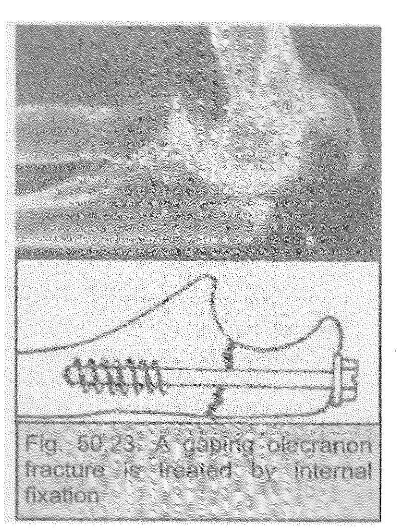


Fig. 50.23. A gaping olecranon fracture is treated by internal fixation

Complications

1. Loss of some movements especially full extension.
2. Ulnar nerve damage is rare.
3. Secondary osteoarthrosis of the elbow.

Fractures of the coronoid process

Avulsion of the coronoid process by the brachialis muscle may occur as an isolated injury or may be associated with posterior dislocation of the elbow (Fig. 50.21). The coronoid is approximated to its bed when the elbow is flexed 30 degrees above right-angle. Reduction is then held by plaster cast for 3 weeks. Occasionally operative repair is needed.

Treatment

1. Undisplaced fractures are treated by a sling till pain decreases and then active movement should be encouraged.
2. Displaced fractures are treated by either excision of the radial head, internal fixation or by conservative measures as the undisplaced variety.
3. Comminuted fractures should be treated by early excision of the whole head to prevent adhesions and joint stiffness.

Fractures of the shafts of radius and ulna

Fracture of one bone without angulation

This is caused by direct trauma. It requires immobilization in an above elbow plaster for 6 weeks in an adult and for 3 weeks in a child.

Fracture of both bones

Trauma and morbid anatomy

- Direct blow will break the bones at approximately the same level (Fig. 50.24). Indirect trauma, e.g., twisting of the forearm, results in oblique fractures situated at different levels in the two bones.
- The fracture may involve the upper, middle or lower thirds of the forearm.
- Besides overlap, angulation and side displacement, rotation may occur due to unbalanced pull of the supinator and pronator muscles attached to the radius.
    - If the fracture occurs above the insertion of the pronator teres, the proximal fragment is pulled into supination by the biceps and supinator muscles.
    - If the fracture is below the insertion of the pronator teres, the proximal fragment is in mid pronation.
    - This is important to bear in mind while planning for reduction and fixation. The distal segment should be brought in alignment with the proximal one.

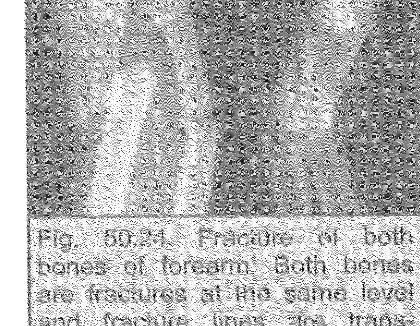


Fig. 50.24. Fracture of both bones of forearm. Both bones are fractures at the same level and fracture lines are transverse. The trauma was likely to be a direct one.
Notice also that this is a child and that the ulnar fracture is a green-stick one where one side of the cortex is intact.

Treatment

1. Undisplaced fractures are treated by a full arm plaster cast with 90° elbow flexion and neutral rotation, for approximately 6-9 weeks.
2. Displaced fractures. In adults it is necessary to achieve perfect reduction, otherwise, loss of pronation and supination will occur. In children residual angulation of about 5° can be accepted as it will be corrected by remodeling.
    a. In adults open reduction and compression plating is done. If internal fixation is not rigid enough it should be supplemented by an above the elbow plaster cast for 6 weeks.
    b. In children closed reduction and plaster fixation is usually successful.

Complications

1. Non-union is rare and results from inadequate fixation or infection.
2. Malunion. Adequate treatment is a must from the start.
3. Nerve injury is uncommon.
4. Acute compartment syndrome may occur and should be treated by fasciotomy.
5. Synostosis (cross-union) between radius and ulna.

Monteggia fracture dislocation

The presence of a fracture of one forearm bone should always raise the suspicion that the inferior or superior radio-ulnar joint has been dislocated as well. This is confirmed if angulation occurs at the fracture site, for this is only possible in a fracture of a single forearm bone if the ligaments connecting the angulating bone to its opposite member have been torn.

Monteggia fracture-dislocation

These consists of fracture of the ulna and dislocation of the superior radioulnar joint.

Types (Fig. 50.25)

1. Extension type 60%. It consists of anterior angulation (apex anterior) of the ulna and anterior dislocation of the radial head.
2. Flexion and lateral types are less common.

Fig. 50.25. Monteggia fracture-dislocation.
1. Extension type.
2. Flexion type.
The X-ray shows the common extension type.

Treatment

Open reduction and compression plating of the ulna and closed reduction of the radial head. Open reduction of the radial head and repair of the annular ligament is only done if closed reduction fails. This is followed by the application of a full-arm plaster cast for 6 weeks.

Complications

1. Posterior interosseous nerve injury.
2. The fracture may be missed in children. The ulna may not fracture. It may just bend enough to allow the radial head to dislocate.

Fractures of the distal radius and ulna

Colles' fracture

Trauma and type of patient

This is a fracture of the distal end of the radius that is produced by a fall on the palm of the outstretched hand. It is not common below the age of 50. After this age it becomes increasingly common particularly in women due to postmenopausal osteoporosis.

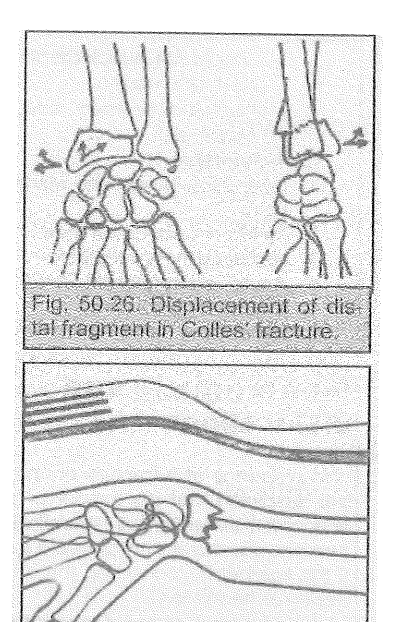


Fig. 50.26. Displacement of distal fragment in Colles' fracture.

Fig. 50.27. Dinner-fork deformity.

Pathological anatomy

The fracture line is within one inch of the lower end of the radius in its cancellous part. It runs obliquely upwards and backwards so that the distal fragment shows the following displacement (Fig. 50.26):

1. Dorsal displacement and tilt.
2. Radial displacement.
3. Upward displacement and impaction.
4. Supination. The fracture is often comminuted especially on the dorsal aspect.

There is always an associated injury to the inferior radioulnar joint and the ulnar styloid process may be avulsed.

Clinical features and diagnosis

- The displacement causes the characteristic 'dinner-fork' deformity (Fig. 50.27) Thus, viewed from the side, the dorsal aspect of the wrist is unduly prominent, while from the dorsum the lateral aspect of the wrist is slightly prominent and the hand is radially deviated.
- The fracture site is tender.

- The radial styloid process is no longer distal to the ulnar styloid; the two are approximately at the same level, a reflection of shortening of the radius.
- X-rays is diagnostic (Fig. 50.28).

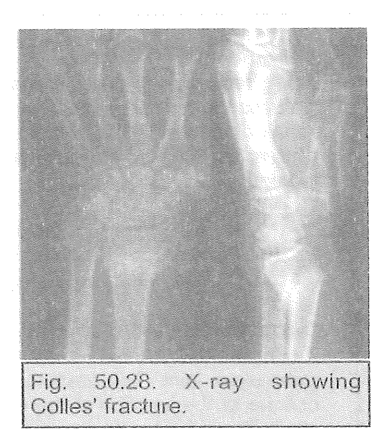


Fig. 50.28. X-ray showing Colles' fracture.

Treatment

- **Reduction**. This is done by manipulation under anaesthesia. An assistant grasps the upper arm while the surgeon grasps the injured hand as if 'shaking hands'. The manipulation is carried out by 3 grips (Fig. 50.29):
    - Traction along the long axis of the limb in order to disimpact the fragments.
    - Pressing the distal segment anteriorly with palmar flexion and pronation to correct the posterior displacement and tilt.
    - Pushing the distal fragment towards the ulna to correct the radial displacement and tilt.
- Fixation. A below elbow plaster cast is applied holding the wrist in palmar flexion and ulnar deviation.
- After care. The circulation to the fingers should be carefully watched in the initial 24 hours for fear of a tight plaster cast.
    - The fingers should be actively mobilized immediately after the injury and the patient should also be instructed to elevate the arm above the head several times daily to prevent stiffness. This is in fact more important than the treatment of the fracture itself in terms of functional outcome.
    - The plaster is maintained for 6 weeks.

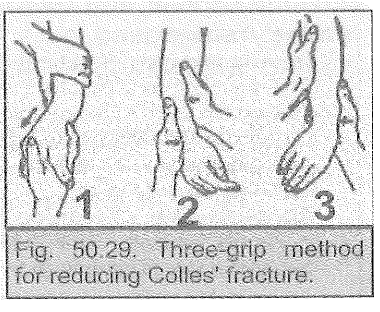
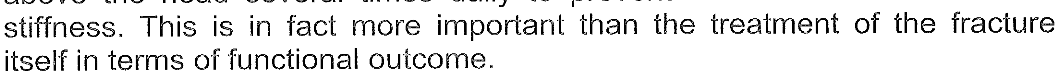


Fig. 50.29. Three-grip method for reducing Colles' fracture.





Complications

1. **Malunion**. While union is the rule, there is often some residual deformity because:
    a. Redisplacement may occur inside the cast especially if the posterior cortex is comminuted.
    b. A Colles' plaster does not control the supination element of the deformity. In most cases exercises and physiotherapy is all that is needed.
2. **Stiffness of the wrist**, shoulder and fingers.
3. **Carpal tunnel syndrome**: This is a late complication which is treated by surgical division of the flexor retinaculum after confirmation of the diagnosis by nerve conduction studies.
4. **Rupture of the extensor** pollicis longus tendon, Late rupture at the level of the fracture may occur. The torn tendon is frayed because of friction over a rough bed.
5. **Sudeck's atrophy** (Chapter 49).

Fractures of the hand

Fracture of the scaphoid

The injury occurs following a fall on the outstretched hand, typically in young adults. The fracture may occur at:

1. The waist of the bone (commonest site) as the bone is bent at its waist over the radial styloid process (Fig. 50.31).
2. The proximal third (proximal pole fracture).
3. The distal tubercle.

Fig. 50.31. The scaphoid commonly fractures at its waist.

Clinical features and radiology

- There is pain in the wrist, but function of the wrist may not be grossly impaired.
- On examination there is tenderness over the scaphoid in the anatomical snuff box (Fig. 50.32), a little swelling and no bruising. These physical signs often appear trivial and suggest a sprained wrist rather than a fracture.
- Radiological examination, including AP, lateral and oblique views, may not be informative immediately after the injury because no displacement occurs at the fracture site.

Fig. 50.32. Scaphoid fracture is commonly mistaken for wrist sprain.

Treatment

Because this fracture does not always produce obvious clinical and radiological signs, it is often missed and wrongly diagnosed as a "sprained wrist". The wrist is then treated with strapping and as a result the fracture may not unite.

Fig. 50.33. A scaphoid plaster cast should extend to involve the proximal phalanx of the thumb.

1. If suspected a scaphoid plaster should be applied for 2 weeks.
2. The wrist is then re-X-rayed. By this time bone resorption should have occurred at the fracture site which should be radiologically evident.
3. The wrist is fixed in a below elbow plaster cast including the proximal phalanx of the thumb (Fig. 50.33) for 8 weeks. This will result in bony union in 90% of uncomplicated cases.

Complications

1. Non-union is treated by bone grafting with or without internal fixation.
2. Avascular necrosis of the proximal fragment may occur because the blood supply to the proximal part of the bone often enters entirely through the distal pole.

Fracture of the phalanges

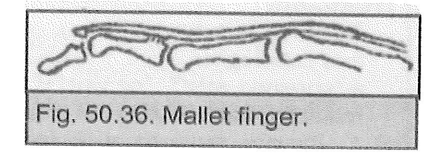


Fig. 50.36. Mallet finger.

These are usually transverse and the fragments are often angulated forwards by the tension of the lumbrical and interosseous muscles. The angulation is corrected and the finger is immobilized in semiflexion over a finger wire splint incorporated in a forearm plaster cast.

Mallet finger (Fig. 50.36)

This is avulsion of the extensor expansion from the base of the terminal phalanx resulting in flexion deformity of that phalanx. The patient cannot actively extend the distal interphalangeal joint. The finger is immobilized with the proximal interphalangeal joint in flexion (so as to relax the lateral slip of the extensor expansion) and the distal interphalangeal joint hyperextended (so as to approximate the end of the tendon to the raw area). Immobilization is maintained for 6 weeks.

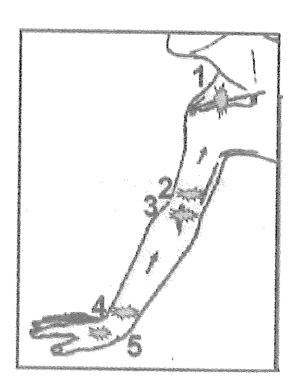


Fig. (50.37): A fall on the outstretched hand produces one of the following common fractures: (1) Clavicle fracture. (2) Supracondylar fracture in children. (3) Posterior elbow dislocation. (4) Colles' fracture in the elderly. (5) Scaphoid fracture.

FRACTURES AND DISLOCATIONS OF THE PELVIS AND LOWER LIMBS

CHAPTER CONTENTS

Fractures of the pelvis

The pelvis is a rigid osseo-ligamentous ring with considerable inherent stability. It consists of the two hip bones and sacrum linked by the very strong sacroiliac, sacrotuberous and sacrospinous ligaments posteriorly and the symphysis pubis and related ligaments anteriorly.

Causative trauma

Fractures of the pelvis usually result from road traffic accidents in persons under the age of 60 years and falls at home in the elderly.

Classification

Figure 51.1 shows the types of pelvic fractures.

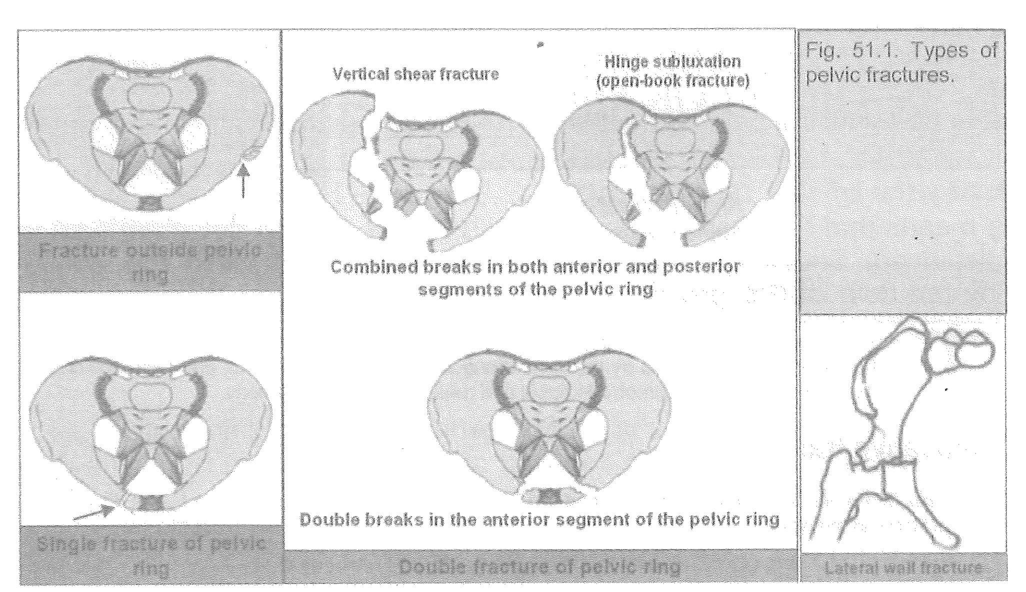


Fig. 51.1. Types of pelvic fractures.

Fractures outside the pelvic ring

Single fracture of the pelvic ring.

Double fractures of the pelvic ring.

- Combined breaks in both the anterior and posterior segments.
- Double breaks in the anterior segment.

Lateral wall fracture

- Acetabular fracture.
- Segmental fracture.

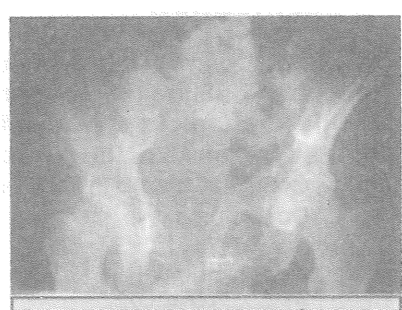


Fig. 51.2. Vertical shear fracture.

Fractures outside the pelvic ring

1. Avulsion injuries of apophyses, e.g., anterior superior and inferior iliac spines, and the ischial tuberosity.
2. Fractures of the iliac wings.
3. Fractures of the sacrum.
4. Fractures of the coccyx.

There are usually no complications and no specific treatment is required other than rest until pain subsides.

Single fracture of the pelvic ring

Trauma and morbid anatomy

- This is usually the result of compression of the pelvic ring either from a crush injury or a force transmitted through the femur to the pelvis (e.g., after a fall).
- The compressed pelvic ring usually gives way at the weakest point; the anterior rami, resulting in a fracture of the pubic or/and ischial rami on the same side. Altematively, the ring may give way at the sacroiliac joint.
- If the pelvic ring is broken in only one place, no significant displacement occurs at the fracture site. clinical features
- Local pain. However, the patient can stand and lift his leg.
- There is bruising and tenderness over the site of injury.
- Springing of the pelvis is painful.

Treatment

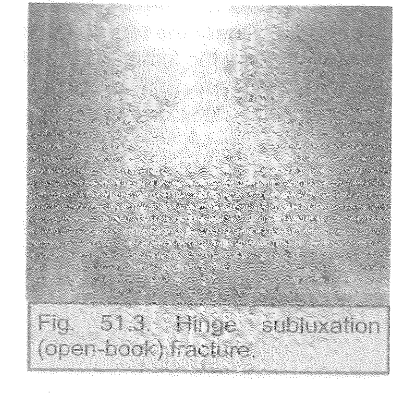
Rest in bed until the pain has settled. The fracture usually unites in about 6 weeks.

Double fracture of the pelvic ring

This means that there are two breaks with an intervening detached segment which may get displaced. Shock is always severe and structures within the pelvis may be damaged by the detached segment. In addition, the pelvis is no longer a stable weight-bearing structure.

Types

1. Combined breaks in both anterior and posterior segments of the pelvic ring
   a. **Vertical shear fracture** consists of fractures of both rami or a disruption of the symphysis pubis at the front, coupled with a fracture of the ilium or sacrum or a disruption of the sacroiliac joint posteriorly (Fig. 51.2). The mobile hemipelvis tends to be pulled upwards by the anterior and posterior trunk muscles. It also opens up like a book by the weight of the leg. This type of pelvic fracture is associated with the highest mortality and morbidity.
   b. **Hinge subluxation** occurs when there is disruption of the symphysis pubis with wide separation in addition to disruption of one sacroiliac joint with little separation (open-book injury, Fig. 51.3). The stability of this injury is variable.


Fig. 51.3. Hinge subluxation (open-book) fracture.

   Both injuries result from either direct anteroposterior or lateral compression from a crushing injury or indirect lateral compression through the lower limb.

2. **Double breaks in the anterior segment of the pelvic ring:** An anterior segmental (tetraramic) fracture usually results from direct violence to the symphyseal region. Fractures of the pubic rami on both sides occur. The detached segment may be displaced backwards. The urethra is often sheared off at the apex of the prostate in a male.

Complications

Of top importance is the liability to severe internal haemorrhage, and injury of pelvic viscera.

1. Posterior urethra in males.
2. Extraperitoneal rupture of the urinary bladder.
3. The rectum.
4. The vagina,
5. Nerve roots.
6. Pelvic arteries and veins. Blood loss may be up to 2.5L

Clinical features

Generally

- The patient is usually shocked due to blood loss inside the pelvis.
- Retroperitoneal haemorrhage may cause ileus and simulates injury to viscera.

Locally

- There is pain in and around the pelvis.
- The patient cannot stand and/or lift his legs, but passive movements at the hips can be elicited.
- One leg may be externally rotated due to opening up of the pelvis and/or short due to upward displacement of the injured side of the pelvis.
- A gap may be felt at the symphysis pubis.

Management

General principles

- This fracture is caused by severe trauma that commonly produces other injuries.
- The principles and priorities of trauma management, which are outlined in chapter 2, should be followed. Correction of the hypovolaemic shock is vital.
- Associated injuries usually take priority over the treatment of bony injuries.

Treatment of the fracture

- **Vertical shear**
    - The aim is to reduce the displacement by applying skeletal traction to the leg to correct leg length and application of a pelvic sling to reduce outward rotation of the hemipelvis. Traction is maintained for 6-8 weeks. Mobilization can now begin, but weight-bearing on the injured side is not allowed for 12 weeks.
    - Open reduction and internal fixation may be needed.
- **Hinge subluxation (open-book fracture)**. The options are:
    - Closed reduction by a pelvic sling.
    - Open reduction and fixation using a plate applied to the superior aspect of the symphysis.
    - An external fixator may be applied to each hemipelvis (for reduction and fixation).
    - No weight bearing for 12 weeks.

- **Tetraramic fracture**
  - Uncomplicated fractures require bed rest for 6 weeks.

Complications of pelvic fractures

1. Shock. Blood loss from a fractured pelvis may amount to 2.5 litres.
2. Visceral injury, e.g., the urinary bladder, urethra, rectum, anal canal, blood vessels and nerves.
3. Paralytic ileus due to retroperitoneal haematoma.
4. Deep vein thrombosis is mainly due to prolonged bed rest.
5. Malunion causes no significant problems except in females (cesarean section may be needed for delivery).
6. Secondary osteoarthrosis commonly occurs after disruptions of the sacroiliac joints and acetabular fractures.

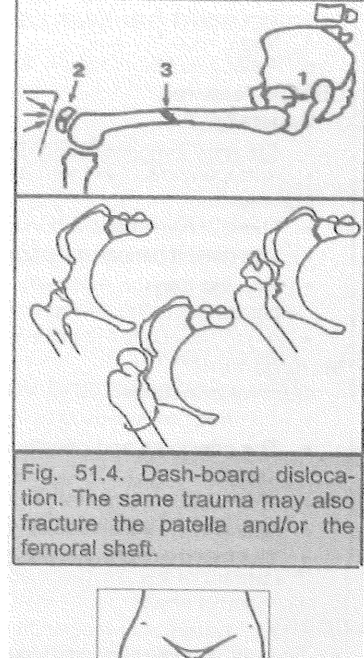
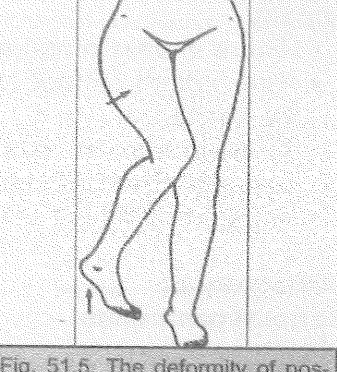


Fig. 51.4. Dash-board dislocation. The same trauma may also fracture the patella and/or the femoral shaft.

Hip dislocation

The hip joint, unlike the shoulder joint, is notable for its stability because of the depth of the acetabular cavity and the strong support afforded by its ligaments and muscles. Therefore, this injury is less frequent. The dislocation may be posterior (by far the commonest), anterior or central.


Fig. 51.5. The deformity of posterior hip dislocation.

Posterior dislocation

Trauma and morbid anatomy

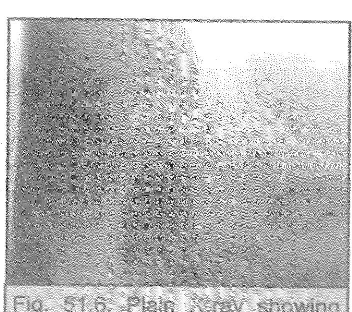
This occurs when the hip is flexed and adducted because in this position the head of the femur is covered posteriorly by the capsule rather than bone. A force applied in the long axis of the femoral shaft may dislocate the head backwards over the posterior lip of the acetabulum. There are two common accidents that produce posterior hip dislocation:

1. A weight falling on the back of a person in a stooping position, as may happen if a coal miner is struck by a "fall of roof'.
2. In car accidents a front seat passenger is thrown forwards so that the knee hits the dashboard "dash-board dislocation" (Fig. 51.4).

The hip commonly dislocates posteriorly, while the shoulder commonly dislocates anteriorly.

Clinical features

- **Severe pain.**
- **Deformity (Fig. 51.5)**
  - The hip is in a position of flexion, adduction and internal rotation.
  - Shortening of the affected limb.
- The greater trochanter is raised, and the femoral head is palpable in the gluteal region.
- Movements of the hip are painful and limited.


Fig. 51.6. Plain X-ray showing posterior hip dislocation.

Radiology (Fig. 51.6)

1. The femoral head lies outside the acetabulum.
2. Shenton's line is interrupted (this is the curve formed by the lower margin of the superior pubic ramus and the lower border of the femoral neck).
3. The lesser trochanter is less apparent because of internal rotation.
4. Association fractures may be detected, e.g., a chip fracture of the posterior wall of the acetabulum.

Treatment

1. Closed reduction is done under general anaesthesia to obtain full muscle relaxation.
2. The patient is placed on the floor and the pelvis is steadied by an assistant.
3. The hip and the knee are flexed, bringing the head of the bone posterior to the acetabulum.
4. The femur is then adducted and pulled vertically upwards so as to draw the head forwards into its socket.
5. The hip is then extended and the reduction is maintained by skin traction for 3 to 6 weeks.

Complications

1. Sciatic nerve injury.
2. Avascular necrosis of the femoral head. The incidence rises with delay in obtaining reduction.
3. Associated fractures. The commonest is a posterior acetabular rim fracture. Others are those of the patella and femoral shaft.
4. Secondary osteoarthritis.

Central dislocation

This is usually the result of a blow on the greater trochanter driving the head of the femur through the floor of the acetabulum. It results in a comminuted fracture of the acetabulum and may be accompanied by other fractures of the pelvis.

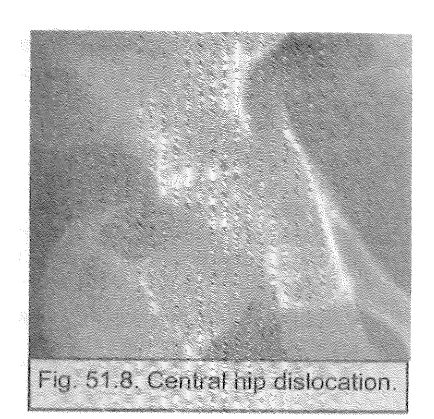


Fig. 51.8. Central hip dislocation.

Treatment

Closed and open methods of reduction are both difficult and unsatisfactory. The best form of management is to accept the displacement and mobilize the hip with tibial skeletal traction as soon as pain allows.

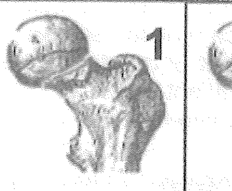
Fractures of the femur

Fractures of the neck of femur

Intracapsular fractures of femoral neck

Pathological anatomy

These occur in that portion of the neck of the femur which lies within the capsule of the hip.



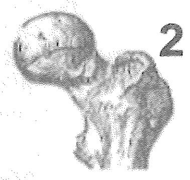


Fig. 51.9. Types of intracapsular femoral neck fractures according to the site:
1. Subcapital.
2. Transcervical.

Classification according to fracture site (Fig. 51.9)

1. Subcapital occurring at the junction of the head and neck.
2. Transcervical occurring somewhere in the neck.

Classification according to displacement

1. Impacted.
2. Unimpacted (displaced).

Trauma

The fracture occurs most commonly in the elderly, especially women, because of senile osteoporosis that weakens the femoral neck. The trauma may be a trivial twisting injury as when the foot catches the edge of a carpet.

Complications

1. **Avascular necrosis**. In intracapsular fracture of the neck of femur, avascular necrosis of the femoral head may occur in 15-35% of cases due to damage of its blood supply. The blood supply of the head of the femur comes from (Fig. 51.10):
   a. An extracapsular arterial ring located at the base of the neck and formed from branches of the medial and lateral circumflex femoral arteries.
   b. Ascending cervical branches of the extracapsular ring lie subsynovially on the surface of the neck. These are known as the retinacular vessels and enter the head by piercing the bone at the junction of head and neck.
   c. Arteries of the ligamentum teres which, in the adult, are not always patent and, if they are, probably only supply a small area of bone.
   d. Terminal branches of the ascending nutrient arteries are present within the neck and share in supplying the head of the femur.

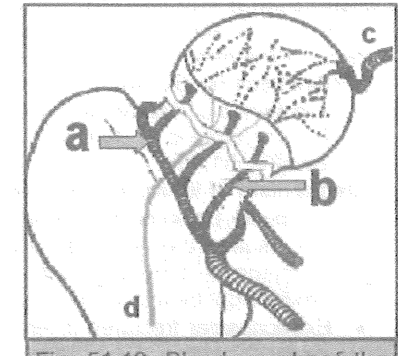


Fig. 51.10. Blood supply of the head of femur.

   Sequelae
   - **Delayed or even non-union:** may occur due to:
     a. Adequate immobilization is difficult to achieve even by internal fixation.
     b. Poor blood supply to the proximal fragment.
   - **Secondary osteoarthrosis**.
2. **Thromboembolism** occurs in 25% of patients.
3. Mortality is approximately 20% during the first 3 months after fracture in the elderly subjects.


Fig. 51.11. Characteristic position of right femoral head fracture.


Fig. 51.12. Displaced subcapital fracture of the neck of right femur. Notice the osteoporosis.

Clinical features

Unimpacted fractures

Inspection

1. The hip is adducted and externally rotated (by the weight of the limb, Fig. 51.11).
2. Shortening of the limb.
3. The patient is unable to lift the leg off the examination couch.

Palpation

1. The hip is tender anteriorly.
2. The greater trochanter is raised.
3. Movements of the hip are painful.

In impacted fractures no rotation occurs and the fracture appears to be abducted rather than adducted. There is tenderness over the fracture site, but otherwise there may be no abnormal physical signs. These fractures are, therefore, easily missed clinically.

Radiography

An exact diagnosis requires X-ray examination (Fig. 51.12). However, even on X-ray examination, impacted fractures with minimal displacement may be missed.

Fig. 51.13. Hemiarthroplasty.

Treatment

Treatment is invariably operative because the proximal fragment can neither by properly manipulated nor immobilized by conservative means. Internal fixation also helps early mobilizatioh of these elderly patients who are prone to the complications of prolonged recumbency.

Fig. 51.14. Types of extracapsular femoral neck fractures according to the site:
1. Trochanteric.
2. Subtrochanteric.

Treatment of impacted fractures

No reduction is required. Internal fixation, preferably with two cannulated screws.

Treatment of displaced fractures

1. **Patient under 65**. Closed reduction followed by internal fixation as above should be undertaken. If a satisfactory closed reduction cannot be obtained, an attempt at open reduction should be performed.
2. **Patient over 65**. The treatment of choice is replacement of the head and neck of the femur with a metal prosthesis (hemiarthroplasty, Fig. 51.13). The reason for this is that the chance of non-union or of avascular necrosis is high.

Postoperative

In all cases, postoperatively, the patient can be allowed out of bed as soon as the general condition allows.

Extracapsular fractures of femoral neck

This term is applied to fractures extending from the base of the neck of the femur to 8 cm below the lesser trochanter. They are divided into (Fig. 51.14):

1. Trochanteric fractures down to the level of lesser trochanter.
2. Subtrochanteric fracture from the lesser trochanter to 8 cm below (Fig. 51.15).



Fig. 51.15. Subtrochanteric fracture.

Mechanism of injury and morbid anatomy

- In young persons trochanteric and subtrochanteric fractures are usually the result of major trauma. In the elderly trochanteric fractures may occur secondary to a fall on the side producing a blow over the greater trochanter.

- Extracapsular fractures differ from intracapsular ones in 2 aspects:
  - The blood supply of the two fragments is good and so avascular necrosis and non-union do not occur.
  - The proximal fragment can be controlled conservatively and so operative treatment is not mandatory.

Clinical features

1. External rotation and shortening of the limb.
2. Tenderness over the fracture site.
3. Inability of the patient to raise his leg.

Complications

1. Thromboembolism.
2. Malunion leading to shortening, adduction and external rotation.

Treatment

- Trochanteric fractures occur most frequently in the elderly and the best treatment is to do internal fixation by a dynamic hip screw (Fig. 51.16). In young persons the fracture may be treated by immobilization and traction to the limb.
- Subtrochanteric fractures are treated by internal fixation by plate and screws.

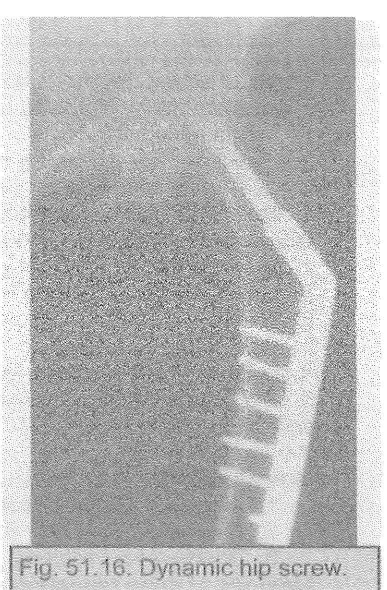


Fig. 51.16. Dynamic hip screw.



Fractures of the shaft of femur

Mechanism of injury

This fracture can occur in children and adults. In young adults, the injury is usually due to severe violence and, therefore, associated injuries are common.

Displacement

- Fractures at the upper third. The proximal fragment is flexed by the iliopsoas, abducted by the gluteal muscles and everted by the external, rotators. The lower fragment is adducted by the adductor muscles, drawn proximally by the hamstrings and everted by the weight of the limb.
- Fractures of the lower two thirds. The distal fragment is posteriorly angulated.

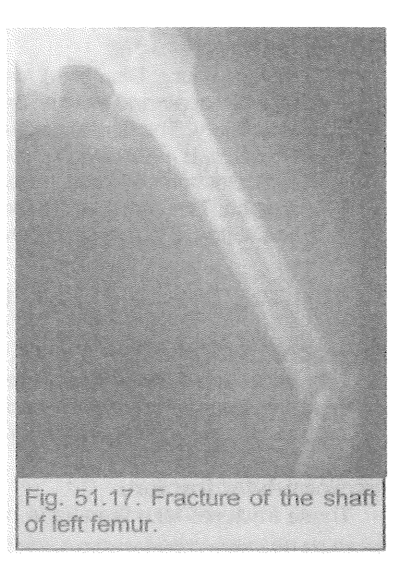


Fig. 51.17. Fracture of the shaft of left femur.

X-ray should be performed to the pelvis and knee to rule out associated injuries. Fig. 51.17 shows an angulated fracture of femoral shaft.

Treatment

Fractures of the shaft of the femur can be treatment by conservative or operative treatment (Fig. 51.18).

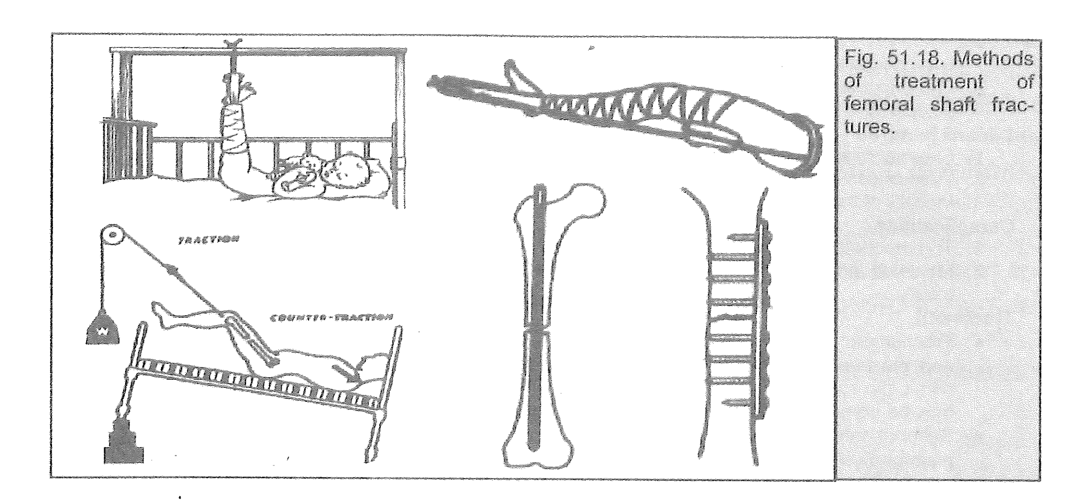


Fig. 51.18. Methods of treatment of femoral shaft fractures.

General principles

1. Proper alignment is important as any mal-alignment will lead to osteoarthrosis of the knee joint.
2. Shortening more than 2 cm should not be allowed. The strong muscles of the thigh tend to cause overriding of the fragments with consequent shortening.

Conservative treatment. The idea is to reduce the fracture and then keep it immobilized by applying traction to the distal segment, and counter-traction to the proximal fragment.

There are two methods for applying this principle:

1. **Sliding traction**. Traction is applied to the distal fragment by weights and pulleys attached to the limb either by skin strapping in children or by skeletal traction in adults by inserting special pins in the upper end of the tibia.
   Counter-traction depends upon the weight of the body acting by elevation of the foot of the bed. The tendency for backward angulation of the two fragments of the femur is controlled by slings under the thigh. The applied weight must be adjusted accurately. Otherwise, distraction of the two fragments occurs with delayed union. The same principle of sliding traction is applied to treat femoral fractures in children below 2 years by using gallows' splints by strapping of the legs and slinging them to a cross piece, so that the pelvis is lifted from the mattress.
2. **Fixed traction by using a Thomas splint**. Cords are attached to the distal fragment and are tied to the foot of the splint. Counter-traction is exerted by pressure of the ring of the Thomas splint against the ischial tuberosity and the soft tissues.

Disadvantages of conservative treatment

1. Prolonged immobilization with all its side effects especially in elderly persons.
2. Liability to stiffness of the knee.
3. It may be impossible to perform reduction if there is soft tissues interposition between the fragments.
4. Difficulty in adjusting the ideal traction; excess traction may lead to distraction.
5. The lateral popliteal nerve may be injured by compression of the side bar of a Thomas splint.

Operative treatment (open reduction and internal fixation). This avoids the disadvantages of conservative treatment.

Indications

1. Inability to perform closed reduction due to interposition of soft tissues.
2. Associated vascular injury, to guard against possible disruption of the vascular repair.
3. Double-level fractures.

> **Supracondylar fractures**
> - Of the femur may injure the popliteal artery.
> - Of the humerus may injure the brachial artery.

Methods of open reduction and internal fixation

1. Fractures of the proximal shaft are fixed by plate and screws.
2. Fractures of the middle two quarters are immobilized by an intramedullary nail.

Complications

1. **Vascular injury**. Displaced fractures of the lower third may injure the femoral or popliteal vessels. Vascular repair and internal fixation should be performed.
2. **Nerve injury**. The lateral popliteal nerve may be injured by the outer bar of a Thomas splint.
3. **Non union is uncommon**. Possible causes include soft tissue interposition, distraction, infection or insufficient immobilization. Treatment is by internal fixation and cancellous bone grafting.
4. **Malunion**. The usual deformities include shortening, varus and external rotation.
5. **Knee stiffness** is a serious complication especially after treatment by closed reduction and traction.

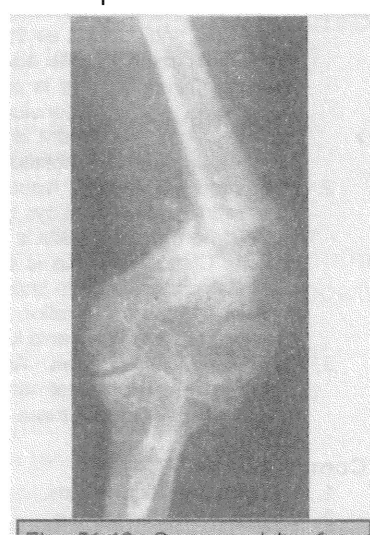


Fig. 51.19. Supracondylar fracture of the femur.

Fractures of the distal femur

Supracondylar fracture (Fig. 51.19)

This fracture is very difficult to treat conservatively as the gastrocnemius muscle flexes the distal fragment, producing posterior angulation at the fracture line. This can possibly injure the popliteal artery. The best results are obtained by internal fixation of the fragments using plate and screws, followed by early mobilization of the knee.

Intracondylar fracture

These fractures take the shape of T or Y fractures, which are similar to those occurring in the distal humerus.

Mechanism of injury

1. Indirect violence. A fall from a height may drive the tibia upwards into the intercondylar fossa.
2. Direct violence due to a blow to the anterior aspect of the flexed knee which drives the patella backwards, splitting the two femoral condyles from the femoral shaft.

Treatment

This fracture is liable to be followed by osteoarthrosis of the knee. Every attempt should be made to reconstruct the joint surface especially in young patients. For them open reduction and internal fixation by screws is the best line of treatment.

Fracture-separation of lower femoral epiphysis

In an adolescent, the lower femoral epiphysis may be displaced.

Complications:

1. The popliteal artery may be injured by the lower femur. The situation is similar to that of supracondylar fractures in adults. There is danger of gangrene unless the hyperextenion injury is reduced without delay.
2. Interference with growth from damage to the growth plate sometimes occurs.

Treatment

Displacement is corrected under general anaesthesia and the position is maintained in plaster of Paris for 6 weeks.

Extensor apparatus injuries

Extensor mechanism injury results when a patient falls on his leg at the same time as his quadriceps tendon is contracting. The precise location of the lesion varies with the patient's age. In the elderly the injury is usually above the patella; in middle life the patella fractures and in young adults the patellar ligament can rupture.

Rupture above the patella

> There is a lot of similarity between fractures of the olecranon and those of the patella.

This injury occurs in elderly, or in patients on long-term corticosteroid treatment. Rupture may occur in the belly of the rectus femoris which retracts and forms a characteristic lump in the thigh. The function is usually good, so no treatment is required except in athletes, where early suture is probably advisable to maintain power. When avulsion of the quadriceps tendon from the upper pole of the patella occurs; operative repair is essential.

Rupture below the patella

This injury occurs in young people. The ligament may rupture or may be avulsed from the lower pole of the patella. Operative repair is necessary.



Fig. 51.20. A displaced transverse fracture of the patella is treated by internal fixation using wires.

Fractures of the patella

Mechanism of injury

1. Indirect injury is due to forced flexion of the knee when the quadriceps muscle is contracting. In this case the fracture is transverse.
2. Direct injury is due to trauma to the anterior aspect of the flexed knee leading to comminuted fractures.

Types of fractures and their treatment

1. **Undisplaced transverse fractures**. The two fragments of the patella are undisplaced as they are held in position by the pre patellar expansion of the quadriceps tendon and patellar retinaculae. Clinically there is pain and tenderness at the site of the fracture. Active extension of the knee is still possible although painful. Treatment is by immobilization of the knee in plaster for 6 weeks combined with quadriceps exercises.
2. **Displaced transverse fractures**. These are due to more severe trauma which destroys the patellar retinaculae. The proximal segment of the patella is

drawn upwards by the quadriceps muscle. Clinically, a gap is palpable in the patella and active extension of the knee is impossible. Treatment is by open reduction and internal fixation (Fig. 51.20). Early physiotherapy after the operation prevents knee stiffness.

3. **Comminuted fractures**. Accurate reduction is impossible. Therefore, the best treatment is patellectomy followed by reconstruction of the soft tissue component of the extensor apparatus.

Complications

1. Secondary osteoarthrosis.
2. Chondromalacia patellae.

Injuries of the knee joint

Meniscus tears

Functions of knee menisci

1. Increase stability of the knee.
2. Control complex rolling and gliding actions of the joint.
3. Distribute load during movement.

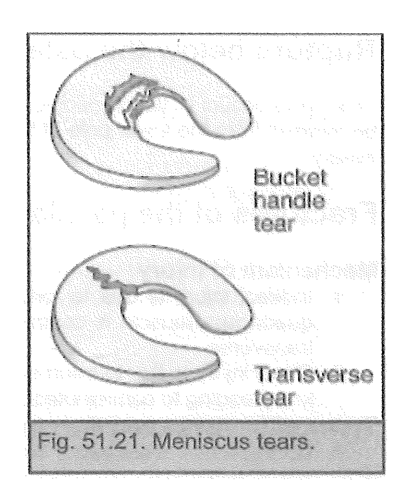


Fig. 51.21. Meniscus tears.

Causative trauma

The meniscus is split by a force that grinds it between the femur and tibia. It commonly affects foot-ball players when weight is being taken on the flexed knee and there is a twisting strain (Fig. 51.21).

Pathological anatomy

- The medial compartment of the knee carries about 90% of the load during weight bearing and the medial meniscus is much less mobile than the lateral, partially because of its attachment to the capsule. Accordingly, meniscus injuries are more frequent on the medial than on the lateral side.
- The tear is usually longitudinal (bucket-handle tear) but may be transverse (parrot-beak tear). A meniscus is avascular and a tear does not heal unless it is peripheral (outer third) which is vascularized from the capsule.

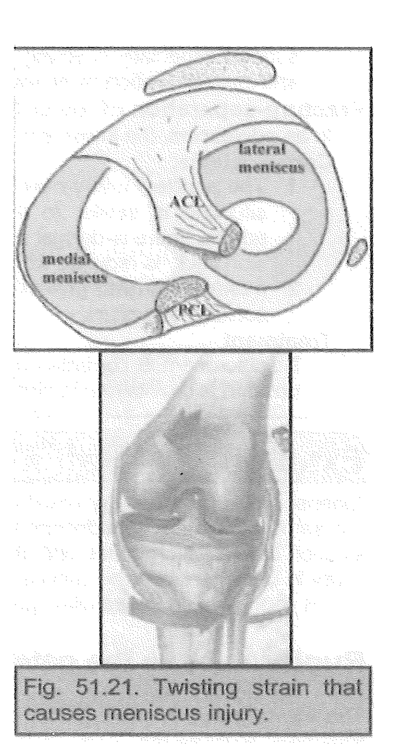


Fig. 51.21. Twisting strain that causes meniscus injury.

Clinical features

History

A typical history is that of injury while playing a game. The player falls, feeling pain in the knee. He cannot resume the game, but can walk home with a limp. The patient may complain of giving way, locking of the joint or recurrent effusion.

Examination

1. Knee effusion.
2. Limitation of complete extension when the knee is locked.
3. Tenderness localized to the joint line on the medial side.

4. Positive McMurray's sign (a click may be elicited when the knee is rotated in certain degree of flexion).
5. Atrophy of the quadriceps muscle is noticed in long-standing cases.

Investigations

- Plain X-rays are normal.
- MRI is the most reliable method and may even reveal tears that are missed by arthroscopy.
- Arthroscopy has the advantage that if a lesion is identified, it can be treated at the same time.

Treatment

Conservative treatment is often acceptable if the knee does not lock.

- Initial treatment is by rest, ice, compression, elevation and non-steroidal anti-inflammatory analgesics.
- The joint is held straight in a posterior slab for 3-4 weeks; quadriceps exercises are encouraged.

Operative treatment

Indications

1. The joint cannot be unlocked.
2. Recurrent symptoms as pain, swelling (effusion) or locking.

Operation

Menisectomy, either by open or arthroscopic technique. The damaged part of the meniscus and loose fragments are removed.

Postoperative quadriceps strengthening exercises.

Knee ligament injuries

Two sets of ligaments in the knee give it stability; the cruciate and the collateral ligaments (Fig. 51.22).

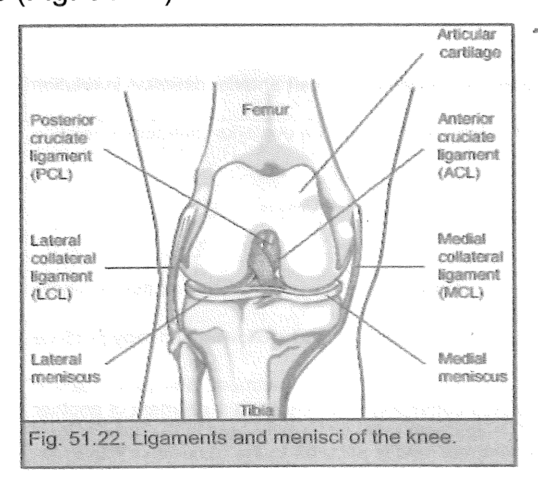


Fig. 51.22. Ligaments and menisci of the knee.

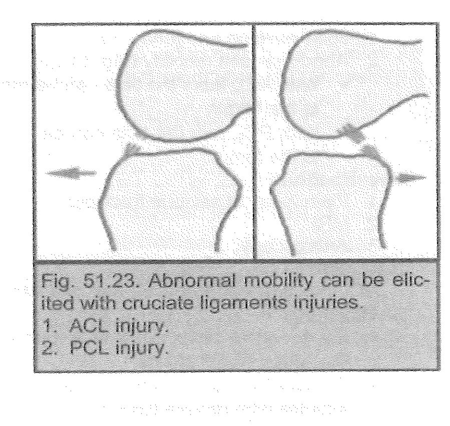


Fig. 51.23. Abnormal mobility can be elicited with cruciate ligaments injuries.
1. ACL injury.
2. PCL injury.

Injuries of cruciate ligaments

The cruciate ligaments are located inside the knee joint and connect the femur to the tibia. They function like short ropes that hold the knee joint tightly in place with flexion and extension. This stability is needed for proper knee joint movement.

The cruciate ligament located toward the front of the knee is the anterior cruciate ligament (ACL), and the one located toward the rear of the knee is called the posterior cruciate ligament (PCL). The ACL prevents excessive sliding of the tibia forwards beneath the femur, while the PCL prevents the opposite movement.

Causative trauma

- The ACL is most often torn by a sudden twisting motion.
- The PCL is not injured as frequently as the ACL. POL sprains usually occur because the ligament was pulled or stretched too far, e.g., by a blow to the front of the knee.

Clinical features

Symptoms

1. Injury to a cruciate ligament may not cause pain.
2. The person may hear a popping sound.
3. The leg may collapse when he tries to stand on it. ACL injuries produce more instability.

Signs

1. Joint swelling and tenderness,
2. Abnormal joint mobility (Fig. 51.23).
   - With ACL tears the tibia can be made to glide excessively in an anterior direction in relation to the femur.
   - With PCL tear the tibia can be made to glide excessively in a posterior direction in relation to the femur.

Investigations

1. Plain X-ray to exclude fractures.
2. MRI.
3. Arthroscopy.

Treatment

ACL injuries are more frequent and are more likely to require surgery because of the instability they produce.

- Conservative treatment similar to that for meniscus injury is initially followed and may produce resolution of symptoms.
- Surgery by reconstruction of ACL is required when conservative treatment fails, particularly in athletes who require full joint function. Surgery commonly involves using a segment of another larger ligament to replace the torn ACL. The ligament most commonly used is the patellar ligament. About one-third of this ligament is removed and is secured to the femur and tibia to replace the torn ACL. It is not possible to repair the torn ACL by simply reconnecting the torn ends. Postoperative physiotherapy is essential.

Injuries of collateral ligaments

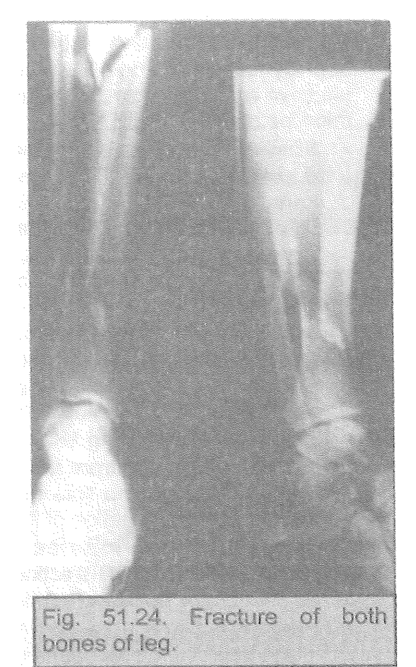
The medial and lateral collateral ligaments may be injured by excessive valgus and varus strains to the knee, respectively.

Pain, swelling, tenderness and excessive valgus or varus movement point to the possibility of this injury. MRI is diagnostic.

Most cases respond well to conservative treatment similar to that for meniscus injury.

Fractures of the shaft of the tibia

Some of these may be isolated fractures of the tibia, while others involve both the tibia and fibula.


Fig. 51.24. Fracture of both bones of leg.

Isolated fractures of the tibia

These are usually due to torsion leading to a spiral fracture with slight displacement. Treatment is usually conservative by an above-knee plaster cast.

Fractures of the tibia and fibula (Fig. 51.24)

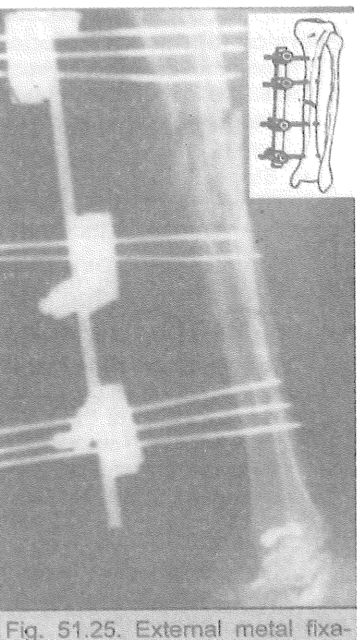
Trauma and morbid anatomy. These fractures occur as a result of direct or indirect violence.

- Direct violence is usually due to road traffic accidents. The fractures occur at the same level in the two bones and are transverse or oblique. They are usually compound and there is often significant displacement.
- Indirect violence may occur due to torsion or bending injury.

Treatment.

One of the following methods may be followed:

1. Closed reduction and plaster fixation is followed in patients under the age of 16, and for fractures in older patients in whom a stable reduction can be obtained.
2. Skeletal traction through the lower end of tibia or os calcis with the leg in a special frame. This method is used if stable reduction cannot be maintained by plaster alone.
3. External skeletal fixation (Fig. 51.25). Special pins are fixed in the tibia above and below the fracture and then attached to special external fixator. This method is very useful for compound fractures of the tibia as it allows for proper reduction and at the same time leaves the wound exposed for dressings, skin grafts or plastic flaps if needed.
4. Internal fixation with a compression plate or an intramedullar nail. This method is used in multiple fractures or in patients in whom reduction cannot be obtained by closed methods.


Fig. 51.25. External metal fixation is suitable for compound fractures of the tibia.

Complications

1. Skin damage. As the shaft of the tibia is directly subcutaneous fractures of the tibia are usually compound. Early closure of the skin by skin flaps is vital for healing of the fracture itself and for prevention of infection. External fixation of the fracture is recommended.
2. Vascular injuries.
3. Delayed and non-union occur in up to 20% of fractures of the tibial shaft due to:
   a. Significant initial injury.
   b. Soft tissue damage.
   c. Infection.
   d. Comminution.

e. Inadequate immobilization.
f. Distraction by excessive traction.

4. Malunion.
5. Infection of compound fractures.
6. Stiffness of the ankle and subtalar joints.

Ankle fractures

Mechanism and morbid anatomy

The ankle is usually injured by indirect violence; the foot being either externally rotated, inverted (adducted), everted (abducted) or, less frequently, internally rotated on the tibia. Rarely, the ankle may be injured by a force which shears the talus transversely relative to the tibia or which drives it vertically up into the tibia.

Although the mechanism is described as if the foot moves on a fixed tibia, in practice, these injuries nearly always occur with the foot fixed, e.g. by a hole in the ground, whilst the tibia continues to move, driven by the momentum of the body weight. These fractures are classified according to the mechanism of injury. Then each type is described in stages; each stage is caused by further continuation of violence.

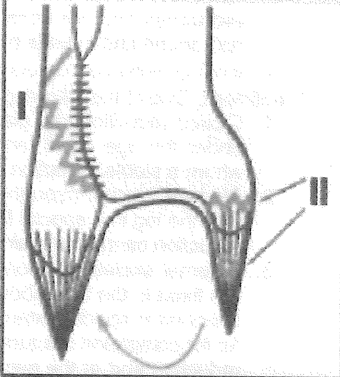
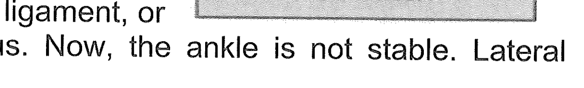


Fig. 51.26. Pott's fracture (external rotation fracture of the ankle).
- **Stage** I. Spiral fracture of the fibula. **Stable fracture.**
- **Stage** II. Deltoid ligament rupture or avulsion fracture of medial malleolus. **Unstable fracture.**
- **Stage III** (not shown) is fracture of the third malleolus. **Unstable fracture.**

External rotation injury (Pott's fracture)

This is the commonest type and is traditionally known as Pott's fracture. During normal walking the feet are placed in a little external rotation relative to the line of travel. A sudden arrest of the weight bearing foot whilst the tibia continues to move forwards has the effect of externally rotating the foot on the tibia and may result in this type of injury (Fig. 51.26).

- **Stage** I. The talus pushes the lateral malleolus posteriorly producing a rupture of the anterior inferior tibiofibular ligament followed by an oblique (spiral fracture) of the distal fibula with a characteristic fracture line which runs downwards and forwards to the level of the ankle joint. The posterior inferior tibio-fibular ligament and the medial structures of the ankle are still intact. Thus the ankle is stable.
- **Stage II.** If the force continues to act, then the medial structures fail either by rupture of the deltoid ligament, or an avulsion fracture of the medial malleolus. Now, the ankle is not stable. Lateral dislocation of the talus may occur.



- **Stage III.** The rotating talus then pushes off a small posterior malleolar fragment from the posterior aspect of the distal articular surface of the tibia (fracture third malleolus). Now, further posterior dislocation of the talus will occur.

Inversion (adduction) injuries

These produce a lateral traction force and medial compression force. The former usually precedes.

- **Stage** I. Rupture of the lateral ligament, or an avulsion fracture of the lateral malleolus at its tip or transverse at the level of the ankle joint. This is stable. No displacement occurs. Sprains and ruptures of the lateral

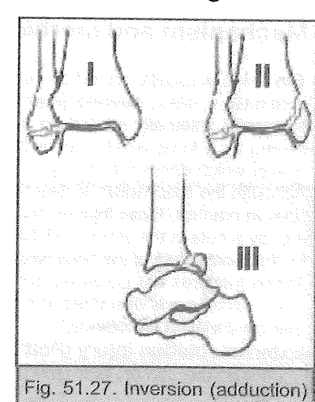


Fig. 51.27. Inversion (adduction) fracture of the ankle.

ligament are the commonest ankle injuries and are usually loosely diagnosed as a 'sprained ankle'. They can only be differentiated from each other by stress radiography.

- **Stage II**. Next, fracture of the medial malleolus occurs with a near vertical fracture line because it is 'pushed off. Medial displacement of the unstable ankle occurs.
- **Stage III**. Rarely, fracture of the third malleolus occurs with further posterior displacement.

Eversion (abduction) injuries

These produce a medial traction force and a lateral compression force. The former usually precedes.

- **Stage I**. Rupture of the deltoid ligament or a transverse fracture of the medial malleolus. The ankle is stable.
- **Stage II**. Next, oblique fracture of the lower fibula extending upwards and outwards from the level of the ankle joint with comminution of the lateral cortex. Lateral displacement then occurs.
- **Stage III**. Rarely, fracture of the third malleolus with further posterior displacement.

Vertical compression injuries

These injuries result from a fall from a height. The pattern of the fracture depends on the position of the ankle at the time of injury.

a. Burst fracture of the ankle. The foot is in neutral position at time of injury. The distal end of the tibia is comminuted and the talus is driven proximally between the tibia and fibula.
b. Anterior marginal fracture of the tibia. The ankle is dorsiflexed at the time of injury. There is often anterior subluxation of the talus. An associated fractured neck o talus may be present.
c. Posterior marginal fracture of the tibia. The ankle is plentar flexed at the time of injury.

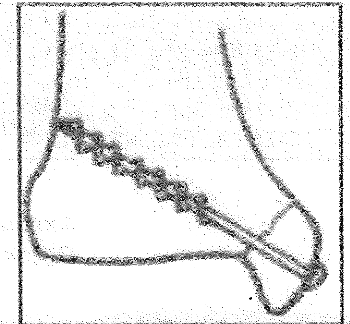




Fig. 51.28. Internal fixation is commonly used for ankle fractures in order to restore a smooth articular surface.

Internal rotation injuries

These are rare and produce injuries similar to those of inversion.

Diagnosis

Clinically, all the signs of a fracture are present. Swelling is particularly severe and deformity is evident, but is variable according to the type of fracture.

Radiography is important. Stress X-rays are very important for detecting ligament tears.

Complications

1. Malunion results from failure to reduce and stabilize unstable fractures.
2. Non-union occasionally occurs in fractures of the medial malleolus probably as a result of in folding of periosteum producing soft tissue interposition. For this reason, this fracture is always best treated by internal fixation.
3. Secondary degenerative osteoarthrosis.
4. Ankle stiffness.

Treatment

Aim. The aim is to restore:

1. The anatomical position of the talus.
2. The joint line parallel to the ground.
3. The smooth articular surface.
4. Stability until the fracture unites by plaster cast or internal fixation.

Guidelines

1. Stable fractures should be treated by application of a weight bearing below-knee plaster cast for 6 weeks.
2. Unstable undisplaced fractures should be treated by application of non weight-bearing plaster and careful follow up with regular check X-rays for 6 weeks.
3. Unstable displaced fractures
   a. In young patients. Accurate open reduction and internal fixation (Fig. 51.28).
   b. In elderly patients. Closed reduction and application of a below-knee plaster cast for 6 weeks.

INFECTIONS OF BONES AND JOINTS

Surgical anatomy of bones

Bone is a specialized form of connective tissue (one third), impregnated with calcium salts (two thirds). The inorganic calcium salts (mainly calcium phosphate and calcium carbonate) make it hard and rigid, which can afford resistance to compressive forces of weight-bearing and impact forces of jumping. The organic connective tissue (collagen fibres) makes it tough and resilient (flexible), which can afford resistance to tensile forces.

Bones have a greater reparative power than any other tissue in the body except blood.

CHAPTER CONTENTS

Classification of bones

According to shape

1. Long bones (femur).
2. Short long bones (carpal and tarsal bones).
3. Flat bones (sternum, ribs, scapula).
4. Irregular bones (vertebra, hip bone).

According to mechanism of ossification

1. Membranous.
2. Cartilagenous.
3. Membrano-cartllgenous.

According to maturity

1. Immature (woven) bone. Collagen is laid down irregularly.
2. Mature (lamellar) bone. Collagen fibres are arranged parallel to each other. This mature bone is of two types:
   a. Compact (cortical) bone.
   b. Cancellous (trabecular) bone which is more porous.
   Table 52.1 compares compact and cancellous bones.

Table 52.1. Differences between cortical and cancellous bone

Cortical bone	Cancellous bone
Forms the shafts of long bones	Forms the vertebrae and the ends of long bones
Metabolic turnover is slow	Metabolic turnover is rapid
It can withstand torsional and bending stresses (contributes to rigidity of bone)	It can withstand compression (contributes to pliability of bone)

Blood supply and relation to infection

Long bones

The blood supply of a long bone is derived from the following sources:

1. Nutrient artery enters the shaft through the nutrient foramen, runs obliquely through the cortex and divides into ascending and descending branches in the medullary cavity. It terminates in the adult metaphysis by anastomosing with the epiphyseal,

metaphyseal and periosteal arteries. It supplies the medullary cavity, inner two thirds of cortex and metaphysis.
2. Periosteal arteries are especially numerous beneath the muscular and ligamentous attachments. They ramify beneath the periosteum to supply the outer third of the cortex.
3. Epiphyseal arteries are derived from periarticular vascular arcades (circulus vasculosus).
4. Metaphyseal arteries are derived from the neighbouring systemic vessels. The metaphysis is the zone of active growth. Before epiphyseal fusion, the metaphysis is richly supplied with blood by end arteries forming hair-pin bends. This is the common site of osteomyelitis in children because, the bacteria or emboli are easily trapped in, causing infarction. After epiphyseal fusion, vascular communications are established between the metaphyseal and epiphyseal arteries. Now the metaphysis contains no more end arteries, and is no longer subject to osteomyelitis.

Short long bones, e.g.; metacarpals and metatarsals

The nutrient artery breaks up into a plexus immediately upon reaching the medullary cavity. Infection, therefore, begins in the middle of the shaft rather than at the metaphysis. In adults, however, the chances of infection are minimized because the nutrient artery is mostly replaced by the periosteal vessels.

Type of bone infections

Acute bone infections

1. Acute haematogenous osteomyelitis.
2. Acute osteomyelitis secondary to open fractures.
3. Acute osteomyelitis in association with orthopaedic implants, e.g., after internal fixation of fractures or joint replacement.

Chronic bone infections

1. Chronic non specific osteomyelitis
   a. Chronic osteomyelitis as a sequel to acute osteomyelitis.
   b. Brodie's abscess.
   c. Chronic sclerosing osteomyelitis (Garre's type).
2. Chronic specific osteomyelitis: Tuberculosis.

Infection may reach the bone and joints via the blood stream from a distant site, or by direct invasion from a skin puncture, operation or an open fracture.

Acute haematogenous osteomyelitis

Acute osteomyelitis is almost invariably a disease of children. It may even affect the neonates, particularly premature babies.

Aetiology

Causative organisms

a. Staphylococcus aureus (80%).
b. Other Gram positive cocci as Streptococcus pyogenes or S. pneumoniae.
c. In children under 4 years of age the Gram negative Haemophilus influenza is a fairly common pathogen.
d. Other Gram negative organisms occasionally cause acute bone infection.

Source Septic foci, e.g., boils or IV lines.
Route Blood

Pathology (Fig. 52.1)

Site

The lesion is almost always in the metaphysis of long bones; most often at the lower end of femur, upper end of tibia and upper end of femur. This predilection for the metaphysis has been attributed to:

1. In children the metaphyses are supplied by end arteries.
2. The metaphysis is the part most liable to trauma.
3. It is the actively growing end of bone.
4. Attachment of ligaments and muscles to the metaphysis makes it the part subject to strain.

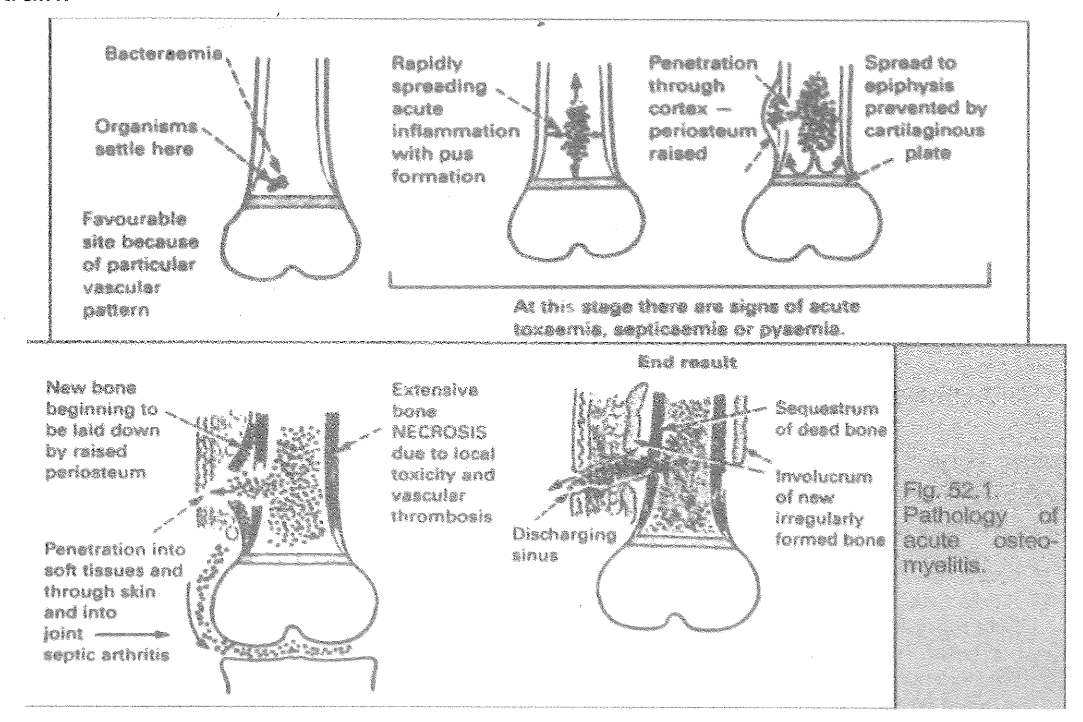


Fig. 52.1. Pathology of acute osteomyelitis.

Natural history

Acute osteomyelitis ends either by resolution or by chronic osteomyelitis. The characteristic pattern of osteomyelitis involves a sequence of inflammation, suppuration, necrosis, reactive new bone formation and ultimately resolution and healing.

1. Inflammation and suppuration. The earliest change is an acute inflammatory reaction with vascular congestion and exudation. By the second or third day, pus forms within the bone and the intraosseous pressure rises rapidly causing intense pain and obstruction of blood flow.
2. Extension and bone necrosis (sequestration).
   - Horizontal spread results in the formation of subperiosteal abscess with resultant obliteration of periosteal vessels and the formation of small cortical sequestra. Later, the abscess may rupture and may open on the skin with resultant discharging sinuses.
   - Vertical extension along the medulla may result in thrombosis of the major nutrient vessels and massive sequestration of the shaft. The epiphyseal cartilage usually prevents spread of infection to the joint. The sequestrum is

dead bone. It looks smooth, shiny white, and devoid from periosteum, and so gives a metallic sound on percussion by a probe. Separation of a sequestrum is known by probing (it moves) and by X-ray where a black hallow is seen surrounding the sequestratum which is dense.

3. **New bone formation forms in two sites.**
   - The deep layers of the stripped periosteum by the end of second week. With time the new bone thickens to form an involucrum enclosing the infected tissue and sequestra. If the infection persists, pus may continue to discharge through perforations (cloaca) in the involucrum and track by sinuses to the skin surface; the condition is now established as a chronic osteomyelitis.
   - Around the Haversian canals resulting in bony sclerosis.
4. **Resolution**. The bone around the zone of infection is at first osteoporotic (probably due to hyperaemia). With healing there is fibrosis and oppositional new bone formation; this together with the periosteal reaction, results in sclerosis and thickening of the bone. Remodeling may restore the normal contours; in others though healing is sound, the bone is left permanently deformed. Chronic osteomyelitis is rare nowadays due to the use of antibiotics and early release of increased intraosseous pressure.

Complications

Systemic

Metastatic infection (pyaemic abscesses).

Local

1. Chronic osteomyelitis.
2. Suppurative arthritis. The epiphyseal cartilage prevents spread of infection to the joint. Suppurative arthritis occurs only in joints where part of the metaphysis is intracapsular, e.g., the shoulder joint.
3. Altered bone growth.
4. Pathological fracture.

Clinical features

Symptoms

- The patient is usually a child with severe limb pain, malaise and fever.
- The child refrains from using the limb.

Signs

- The earliest sign is severe localized tenderness over the inflamed part of the bone.
- Later, there is warmth, oedema and the skin is red.
- Although any movement of the limb is painful, the neighbouring joint can, with gentleness, be moved passively.
- There may be an associated effusion.

Investigations

1. There is polymorphnuclear leukocytosis and raised ESR.
2. A blood sample for culture should be obtained before starting antibiotic therapy.
3. X-ray is normal during the first couple of weeks. Rarefaction and periosteal new bone formation are late radiological findings and treatment should not be delayed while waiting for these to appear.
4. Radioscintigraphy with ^{99m}TcHDP (hydroxymethylene diphosphonate) reveals increased activity in the very early stage, but it has relatively low specificity,

5. The most certain way to confirm the clinical diagnosis is to aspirate pus from the metaphyseal subpierosteal abscess or the adjacent joint.
6. MRI can detect osteomyelitis before the appearance of radiological changes in plain radiography.

Differential diagnosis

1. Cellulitis (in osteomyelitis tenderness is elicited along the bone away from the area of redness).
2. Acute suppurative arthritis. Tenderness is over the joint and all movements (active and passive) are painful and limited.
3. Rheumatic arthritis. The pain tends to be fleeting from one joint to another and there may be carditis.
4. Haemarthrosis.
5. Ewing's sacroma.

Treatment

1. **Antibiotic therapy**. The core of treatment is early administration of high-dose IV antibiotics that are effective against staphylococci. After taking a blood sample for culture start treatment by flucloxacillin at a daily dosage of 200 mg/kg in divided doses. If the patient is sensitive to flucloxacillin, fusidic acid or a course of second generation cephalosporin and gentamycin can be used. When the condition of the patient improves oral antibiotics are prescribed and are continued for 3-6 weeks.
2. Supportive treatment for pain and dehydration.
3. Splinting of the affected part. Splinting is continued until the inflammation subsides and the X-ray shows that there is no risk of a pathological fracture. Full weight bearing is usually possible after 3-4 weeks.
4. Surgical drainage. If there is no good response to antibiotics within 2 days, this denotes the presence of pus which should be drained. Under general anaesthesia and under a tourniquet, the inflamed area is explored, the periosteum is opened to evacuate the pus, which is sent for culture and sensitivity. Some surgeons advise drilling of the cortex to evacuate any pus in the medullary cavity. The wound of the skin is loosely closed.

Chronic non-specific osteomyelitis

Chronic osteomyelitis as a sequel to acute osteomyelitis

The causative organisms are usually a mixed infection (Staphylococcus aureus, E. coli, Strept. pyogenes, Proteus and Pseudomonas). Nowadays, chronic osteomyelitis more frequently follows an open fracture or operation; in the presence of foreign body (implants) the causative organism is usually Staph. Epidermidis, which is normally non pathogenic.

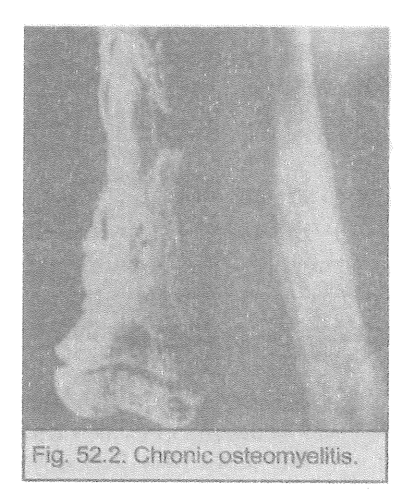


Fig. 52.2. Chronic osteomyelitis.

Pathology

Factors that may maintain chronicity

1. Bone cavity surrounded by dense sclerosis.
2. Sequestrum, which acts as an irritant and harbours bacteria.
3. Bacteria are imprisoned in the fibrous tissue where they remain dormant, and may be activated at any time.

4. Sinuses, which lead to the skin surface favouring secondary infection.

Complications

1. Acute exacerbation.
2. Pathological fracture.
3. Retardation or arrest of growth when the epiphyseal cartilage is destroyed by the disease.
4. Toxaemia and amyloidosis.

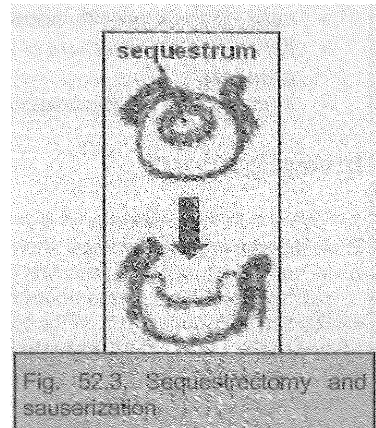


Fig. 52.3. Sequestrectomy and sauserization.

Clinical features

1. History of acute osteomyelitis may be given.
2. The commonest presentation is a sinus which is discharging pus and sometimes small pieces of sequestra.
3. Pain and swelling in the affected bone.
4. There may be atrophy of the surrounding muscles.
5. On examination, the affected bone is thickened, tender with sinuses that are lined by red granulation tissues, exuding pus and when the sinus is probed, the probe will reach the bone.

Investigations

1. **Plain X-ray** (Fig. 52.2) is the most important. It reveals that the bone is thickened with patchy and irregular sclerosis surrounding a bone cavity that may contains sequestra. The latter appears as dense white loose fragments with irregular but sharply demarcated edges.
2. **Radioisotope scanning**. Using technetium (^{99m}Tc)-HDP scans, gallium (7Ga) citrate and indium (^{111}In)-labelled leukocytes; are useful for showing up hidden foci of infection.
3. CT and MRI are valuable in planning operative treatment. Together they will show the extent of bone destruction and reactive oedema, hidden abscesses and sequestra.
4. During acute flares the ESR and blood white cell count may be increased.


Fig. 52.4. Brodie's abscess of lower ulnar metaphysic. Notice the cavity and the surrounding dense sclerosis.

Treatment

Surgery for removal of dead bone and drainage of the cavity is the main line of treatment. Antibiotics alone are of little value because they cannot reach the bacteria hidden in avascular tissue and sequestra. They are of benefit in an acute exacerbation and when given with operative drainage, which often implies:

1. Sequestrectomy.
2. Saucerization of a bone cavity to provide adequate drainage (Fig. 52.3).
3. Obliteration of the dead space by cancellous bone chips or local muscle flaps.
4. Excision of the sinus tract.

Postoperative support of the limb to avoid a pathological fracture until healthy strong bone regenerates.

Brodie's abscess

Pathology

- This commonly affects adolescents and sometimes adults with high resistance resulting in localization of infection that is caused by Staph. aureus or albus.
- A chronic bone abscess is usually of small size, situated in the metaphysis of long bone, commonly the tibia (upper end).
- The abscess cavity may be sterile.
- The abscess is surrounded by dense sclerosis and there is always an associated periosteal reaction.

Clinical features

- Recurrent attacks of pain in the affected area.
- Examination reveals localized swelling and tenderness over the bone and effusion may be present in the adjacent joint.

Investigations

X-ray shows a translucent area surrounded by sclerosis (Fig. 52.4).

Treatment

Surgery. Under antibiotic coverage the cavity is unroofed, pus is evacuated; then curettage and packing with cancellous bone chips if the cavity is not small.

Tuberculosis of bones

Pathology

Tuberculous **osteomyelitis** is always secondary. Mycobacterium tuberculosis is always carried by blood stream from a primary focus elsewhere in the body.



Fig. 52.5. TB osteomyelitis. Notice:
1. Bone destruction without new bone formation.
2. Invasion of epiphyseal cartilage is possible and leads to TB arthritis.

Common sites

The commonest site of TB osteomyelitis is the spine (Potts disease, chapter 57). This is followed by the ribs, stemum, pelvis, tibia, femur and the short long bones of hands and feet.

Special features of TB osteomyelitis

1. Bone destruction and rarefaction predominate over new bone formation,
2. Caseation results in cold abscess and sinus formation.
3. Healing occurs by fibrosis, sequestration is unusual.
4. Tuberculosis can destroy the epiphyseal cartilage plate and eventually involve the neighbouring joint (unlike pyogenic Osteomyelitis).

Complications

1. Cold abscess formation.
2. Involvement of a joint.
3. Sinus formation.
4. General tuberculous toxaemia, miliary TB and amyloid disease.

The commonest site of bone and joint tuberculosis is the spine, followed by the hip joint.

Clinical features

1. Tuberculous osteomyelitis is uaually a disease of children.
2. General manifestations of tuberculosis in the form of malaise, pallor, weakness, loss of appetite and weight, night fever and night sweating.
3. Local manifestations in the form of pain of insidious onset, aching in nature, and more at night. There is a swelling near the joint with marked muscle atrophy. There may be a cold abscess or sinus formation.

Investigations

1. ESR is elevated, blood picture shows anaemia, leucopenia and relative lymphocytosis. Tuberculin test is positive.
2. X-ray. There is localized bone destruction with widespread rarefaction with no new bone formation (Fig. 52.5).

Treatment

1. Improvement of the nutritional state of the patient.
2. Antituberculous drugs. Triple therapy consisting of Rifampicin 10 mg/kg wt, Isoniazid 5 mg/kg wt mg and Ethambutol 25 mg/kg wt/day are given orally for at least 2 to 3 months and thereafter Rifampicin and Isoniazid for a further 6-12 months.
3. Immobilization. Fixation of the limb in position of function in a splint or plaster is performed for up to 6 months where clinical, laboratory and radiological evidence of healing have occurred.
4. Operative treatment. If there is a cold abscess or if conservative treatment failed, operative drainage and curettage are to be performed. In certain sites like tuberculosis of the rib, resection of the rib may be performed.

Other forms of bone tuberculosis

1. Tuberculosis of flat bones (periostitis). The surface of the bone (ribs, sternum, scapula, adult spine or facial bones) is eroded and abscess forms which often opens on the surface with sinus formation.
2. Tuberculosis of short long bones [TB dactylitis, (Fig. 52.6)]. Metatarsals, metacarpals and phalanges are involved and the infection starts in the diaphysis. There is central destruction, and the periosteum is raised by tuberculous granulation tissues, with subperiosteal new bone formation. The bone thus becomes fusiform in shape (spina ventosa).



Fig. 52.6. TB dactylitis (spina ventosa).

Septic arthritis

Acute suppurative arthritis like osteomyelitis is common in children.

Aetiology

Causative organisms

- The usual organism is Staphylococcus aureus.
- In children below 3 years of age, it is mainly due to haemophilus influenza.

Routes of infection

1. Following joint punctures.

2. Direct extension from a nearby infection such as osteomyelitis. This happens if the metaphysis is intracapsular, e.g., the shoulder joint. Otherwise the epiphyseal cartilage prevents spread to the joint.
3. By haematogenous spread from a distant site.

Pathology

Septic arthritis is commonly encountered in the knee and the hip joints; yet any joint may be infected.

1. The organisms first settle in the synovial membrane (synovitis), which becomes red, swollen and exudes pus.
2. The production of chondrolytic enzymes by the infecting organism results in articular cartilage destruction with exposure of the bone ends.
3. Pus may burst out of the joint to form abscesses and sinuses.
4. Later with healing, the raw articular surfaces may adhere, producing fibrous or bony ankylosis.

Recovery of joint mobility can be achieved if treatment begins early at the stage of synovitis before damage of the articular cartilage. Therefore septic arthritis is considered a surgical emergency.

Complications

1. Chronicity.
2. Pathological dislocation.
3. Secondary osteoarthrosis.
4. Pyaemia.

Clinical features

- Suppurative arthritis is more frequent in childhood.
- There may have been a wound.
- Within a few days the patient develops general manifestations of toxaemia with severe throbbing pain and swelling of the affected joint.
- There is deformity, redness, tenderness and limitation of joint movements equally in all directions. Muscle spasm keeps the joint in the position of greatest ease. This position is variable from one joint to another depending on the relative strength of antagonistic muscle groups

Investigations

- There is high leukocytosis and elevated ESR.
- Aspiration of the joint reveals a fluid, which is rich in pus cells. Culture and sensitivity test would reveal the causative organism.
- X-ray. In the early cases (first 2-3 weeks) no radiological abnormalities could be detected. Later, there is rarefaction of the articular ends of the bones followed by narrowing of joint space, irregularity of joint surfaces and subchondral sclerosis.

Differential diagnosis

1. **Acute osteomyelitis**. The tenderness is maximal over the bone not the joint and a small range of joint movement is permitted.
2. Rheumatic fever is polyarticular and fleeting with less severe general manifestations. The condition is relieved by large doses of salicylates.
3. Haemarthrosis. There is a history of trauma and aspiration reveals blood.

4. Tuberculous arthritis. The onset is insidious and is chronic. A cold abscess may be present. Muscle atrophy is marked. X-ray shows rarefaction of bone ends.

Treatment

Septic arthritis is a surgical emergency.

- Treatment of septic arthritis follows the general rule that the treatment of pus anywhere in the body is prompt drainage. Drainage is either arthroscopic or by open arthrotomy.
- IV antibiotics and splinting until inflammation subsides. Later gradual physiotherapy starts.
- If the articular cartilage has been destroyed, the joint is immobilized in the optimum position of function until ankylosis is sound.

Tuberculosis of joints

Pathology

- Joints commonly affected are the hip and knee; but no joint is immune.
- Infection reaches the joint by haematogenous spread from a latent focus from the intestine or the lung or by direct spread from a focus in the adjacent bone.
- The site of initial lesion may be synovial, which is more common in children and in the knee; or osseous where a bone focus may open into the joint.
- Whatever the site of initial lesion, in time, all joint components become affected in the untreated case.
- The synovial membrane is thickened with numerous tubercles, which may form a caseous mass. A synovial pannus would form resulting in cartilage damage.
- Subchondral bone is invaded by granulation tissue. There is marked osteoporosis and bone destruction without new bone formation.
- The surrounding muscles are markedly atrophied.
- Resolution with restoration of complete joint movement never occurs in tuberculous arthritis, which always proceeds to complete destruction of the joint with any of the following sequelae:
    - Healing with fibrous ankylosis.
    - Cold abscess formation.
    - Pathological dislocation.
    - Generalized dissemination.
    - Amyloid disease.

Clinical features

The patient is usually a child or young adult.

Symptoms

- Pain is the earliest symptom.
- The child is unwilling to move the joint and muscle spasm keeps the joint in a fixed position. During sleep muscle spasm lessens and as soon as the joint moves a little the child wakes up crying. This is called night starts or cries.
- General manifestations of tuberculosis; low-grade night fever and sweating, anorexia, loss of weight and weakness.

Signs

- Swelling results from thickening of synovial membrane and rarely effusion.
- Muscle wasting is characteristic.
- Joint movements are limited in all directions.

- As articular erosion progresses the joint becomes stiff and severely deformed.
- In late cases there may be a cold abscess and sinus formation.

Investigations

X-ray shows soft tissue swelling, considerable rarefaction of the bone on both sides of the joint, narrowing of the joint space and blurring of the joint line from erosion of subchondral bone.

Laboratory investigations

1. There is anaemia, leucopenia with relative lymphocytosis, and elevated ESR.
2. Tuberculin test is positive.
3. Synovial fluid assessment reveals low or absent sugar, elevated white cell count and poor mucin precipitate. Bacteriological assessment may reveal tubercle bacilli.
4. In obscure cases synovial membrane or regional lymph node biopsy may reveal the diagnosis.

Treatment

Early cases

As mentioned earlier the mainstay in treatment is antituberculous chemotherapy, using a combination of drugs for 9 months or more. If started early, the joint may heal and function be completely restored. Local measures include rest, traction and occasionally operation. Splintage should be continued for several months, by which time it is usually clear whether the joint has been saved.

Late cases

If the articular surfaces are destroyed, the joint is immobilized until all signs of disease activity have disappeared; then the joint is arthrodesed in the position of maximum function.

- **Always keep in mind the possibility of acute osteomyelitis and septic arthritis in children. They may even affect neonates.**
- **Both conditions require urgent treatment.**
- **For acute osteomyelitis give high-dose intravenous antibiotics. Get samples for blood culture before starting antibiotics.**
- **For septic arthritis emergency drainage is required.**
- **Tuberculosis of bones and joints is a chronic disease.**

GENERALIZED DISORDERS OF BONES AND JOINTS

Bone dysplasias

Bone dysplasias or faulty development, means a generalized disorder of bone and cartilage. These disorders are uncommon and the diagnosis is often difficult. Most dysplasias are familial and patients are short enough to be categorized as dwarfs (less than 125 cm). Metaphyseal aclasis (hereditary multiple exostoses) will be mentioned.

CHAPTER CONTENTS

Metaphyseal aclasis (Hereditary multiple exostoses)

This is the most common of all dysplasias.

Pathology

- Metaphyseal aclasis is characterized by the appearance of multiple exostoses at the metaphyses as the child grows.
- The disease is inherited as autosomal dominant.
- Each exostosis is covered by cartilaginous cap (osteochondromata).
- An exostosis grows only while the child grows and any enlargement after that may herald malignant change to a chondrosarcoma.

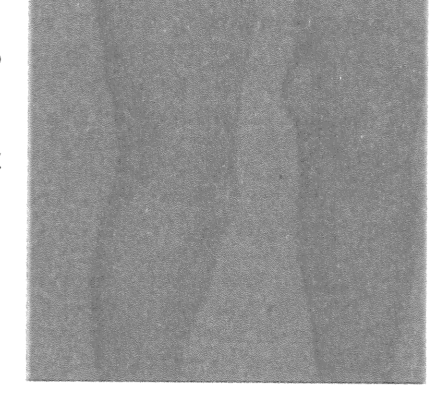


Fig. (53.1): Metaphyseal aclasis

Clinical features

1. Usually the only complaint of the patient is swellings due to the osteochondromata (Fig. 53.1).
2. The child may be slightly short with mild or moderate bowing of the arms or legs.
3. The bony prominences may cause pressure to a neighbouring nerve or blood vessels, or may interfere with the movement of a joint.

X-ray shows the pathognomonic exostoses as well as broadening of the metaphyses.
Treatment: Excision of the bony prominence.

Osteoporosis

Osteoporosis is characterized by a reduction in the amount of bone substance in the skeleton.

Aetiology

Primary (idiopathic) osteoporosis is a public health problem that affects a large sector of the aging population. Risk factors include:

- Old age
- Female
- Sedentary lifestyle
- Menopause before age 45
- Tobacco and alcohol excess

- Fair skin

Secondary osteoporosis

- Hyperparathyroidism
- Cushing's syndrome.
- Calcium, vitamin D, or vitamin C deficiency
- Hyperthyroidism.
- Long-term steroid therapy

Pathology

- Normally the bone mass diminishes slowly but steadily from the age of 40 years. Around the menopause this process is markedly accelerated.
- In osteoporosis the amount of bone substance shows generalized reduction in the whole skeleton.
- The result is reduced bone density which makes them vulnerable to fractures after trivial trauma.
- Histologically, bone trabeculae appear thin with wide marrow spaces.
- There are no demonstrable metabolic changes.

Clinical features

- The disease itself is usually symptomless.
- Osteoporosis constitutes a clinical problem when a fracture occurs. Fractures commonly associated with senile osteoporosis include Colles' fracture, fractures of the vertebral bodies, and fracture of the femoral neck.
- Repeated minor fractures of the spine cause chronic back pain and kyphoscoliosis.

Investigations

- The most widely used techniques of assessing bone mineral density is dual-energy x-ray absorptiometry (DXA).
- Plain X-ray is not sensitive, It shows lessened bone density when bones lose 40-50% of their substance.

Prevention

1. Balanced diet.
2. Calcium and vitamin D supplements for menopausal women.
3. Regular exercise, e.g., walking.
4. Avoidance of alcohol and smoking

Treatment

Secondary osteoporosis

Treatment of the cause, e.g. hyperparathyroidism, hyperthyroidism or hyper-corticism.

Primary osteoporosis

1. Calcium 1500mg/day.
2. Vitamin D 400 U/day.
3. Flouride
4. Calcitonin reduces bone resorption by reducing osteoclastic activity.
5. Biphosphonates also inhibit osteoclastic activity.
6. Hormone replacement therapy for menopausal women.

Complications

Fractures are treated according to their locations and types.

Hyperparathyroidism

This subjected is discussed in detail in chapter 26. Concerning the skeletal system, there is a general loss of bone substance. In more severe cases, osteoclastic hyperactivity produces subperiosteal erosions, endosteal cavitation and replacement of the marrow spaces by vascular granulations and fibrous tissue (osteitis fibrosa cystica).

Haemorrhage and giant cell reaction within the fibrous stroma may give rise to brownish tumor like masses, whose liquefaction leads to fluid-filled cysts (brown tumours).

Clinically the patient complains of bone aches, pathological fractures and deformities.

The earliest X-ray change is resorption of the lamina dura of the tooth sockets and subperiosteal erosions of the cortical bones in the phalanges. There is generalized osteoporosis with thin cortex, deformity and probably bone cysts.

Osteorthrosis (osteoarthritis)

Osteoarthrosis is a degenerative joint disease in which there is progressive loss of articular cartilage accompanied by new bone formation and capsular fibrosis.

Aetiology

1. Primary osteoarthrosis. No cause is obvious.
2. Secondary osteoarthrosis follows a demonstrable abnormality such as trauma, deformity, avascular necrosis (Perthes' disease) or previous joint disease (septic arthritis or tuberculous arthritis).

Pathology

- Osteoarthrosis results from a disparity between the stresses applied to articular cartilage and the ability of the cartilage to withstand such stresses.
- Structural damage follows. The cardinal features are:
    - Progressive cartilage destruction (Fig. 53.2). The cartilage surface is roughened (fibrillation). This extends progressively into deeper tissue until bone is exposed (ulceration).
    - Subarticular cyst formation and sclerosis. Collapse of bone separating such cysts from the synovial cavity results in progressive deformation of bony articular surfaces and the separation of loose fragments of bone.
    - Remodeling of the bone ends and osteophytes formation at the unstressed areas.
    - Capsular fibrosis as a result of low grade inflammatory response induced by loose bodies attached to synovial membrane. This may account for joint stiffness and pain.

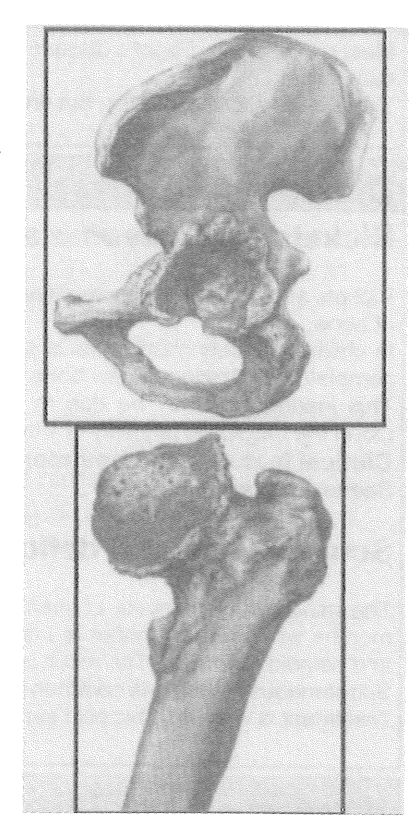


Fig. (53.2): Articular cartilage destruction is the essential feature of osteoarthrosis

Clinical features

Symptoms

- Patients usually present after middle age.
- Pain and stiffness are the main symptoms.
    - Pain is worse on movement and by change of weather.
    - Stiffness is most marked in the morning and gets less by the end of the day.
- Symptoms typically follow an intermittent course, with periods of remission.
- The disease particularly affects the weight-bearing joints such as the hip and knee.

Signs

Examination may reveal:

- Joint swelling due to effusion and thickened synovial membrane.
- Tender joint line.
- Restricted range of movement.
- Deformity.
- Coarse crepitus detected during movement.

Investigations

Plain X-ray reveals the characteristic changes (Fig. 53.3).

- Narrowing of the joint space.
- Subarticular sclerosis.
- Bone cysts.
- Marginal osteophytes.

Sclerosis and osteophytes distinguish this disease radiologically from rheumatoid arthritis, in which the predominant radiological feature is bone loss.



Fig. (53.3): X-ray showing hip osteoarthrosis with narrowing of joint space and subarticular sclerosis

Treatment

Conservative treatment

In the early stages conservative treatment is advised:

1. Load reduction can be achieved by a walking stick, the avoidance of prolonged stressful activity and by weight reduction.
2. Pain relief by analgesics and anti-inflammatory agents.
3. Joint mobility can often be improved by physiotherapy to overcome contractures and to strengthen surrounding muscles.

Surgical treatment

Total joint replacement is indicated when conservative treatment fails to control the symptoms (Fig. 53.4). Replacement of the hip and knee are now common procedures that allow patients to regain painless joint movement.

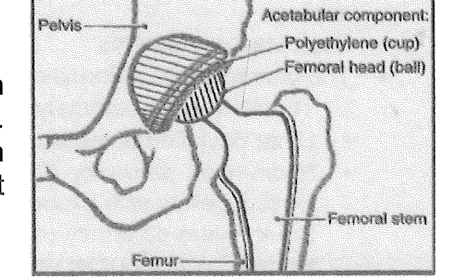


Fig. (53.4): Total hip replacement

Charcot's osteoarthropathy (neuropathic arthritis)

Pathology

- This is a rapidly progressive degeneration in a joint which lacks deep sensations.
- The affected joint is severely disorganized after repeated minor injuries.

- In the lower limb it is associated with tabes dorsalis, cauda equina and peripheral neuropathy, e.g., diabetes. In the upper limb the cause could be syringomyelia (the shoulder) or leprosy (the wrist). The commonest site, however, is Charcot's joint of the foot in diabetics.

Clinical features

There is painless swelling and deformity of the joint (Fig. 53.5). There is effusion, synovial thickening and loose bodies. The patient complains of weakness, instability and abnormal mobility can be elicited. Signs of accompanying neuropathy are present, as anaesthesia, lost tendon jerks and penetrating ulcers of the foot.

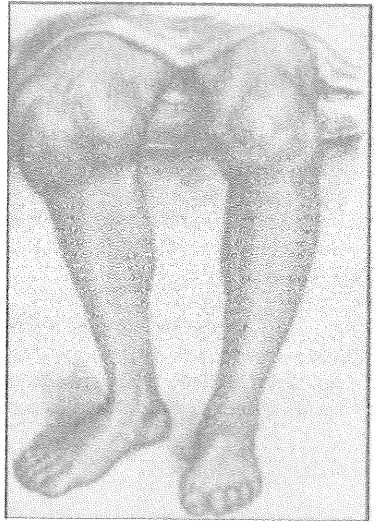


Fig. (53.5): Charcot's knee joint in a case of tabes dorsalis

Investigations

X-ray (Fig. 53.6). There is gross bone destruction, widening of joint spaces and the joint is often subluxated or dislocated. There are irregular calcified masses in the capsule.

Treatment

The underlying condition may need treatment, but the affected joints cannot recover. They should, if possible, be stabilized by external splintage. Surgery is usually not advisable. Amputation may be indicated in Charcot's joints of the feet.

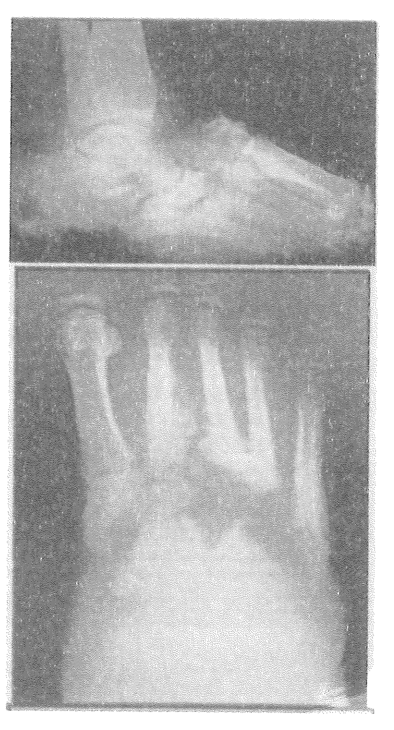


Fig. (53.6): Charcot's joint of the feet in diabetics

BONE TUMOURS

General principles

Tumours, tumour-like conditions and cysts are considered together because their clinical presentation and management are similar. All primary tumours of bone are rare; by contrast, metastatic deposits, especially in those over the age of 50 years, are relatively common. Primary tumours are usually classified by cell type (Table 54.1).

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Table (54.1): Classification of primary bone tumours

Cell type	Benign	Malignant
Bone	Ivory osteoma Osteoid osteoma Osteoblastoma	Osteosarcoma
Cartilage	Chondroma Osteochondroma Chondroblastoma Chondromyxoid fibroma	Chondrosarcoma
Fibrous tissue	Non-ossisfying fibroma (fibrous cortical defect)	Fibrosarcoma
Marrow	Eosinophilic granuloma	Ewing's sarcoma Myeloma Reticulum cell sarcoma
Vascular	Haemangioma	Angiosarcoma
Uncertain	Giant cell tumour	Malignant giant cell tumour

Clinical features

- Age may be a useful clue. Many benign lesions present during childhood and adolescence, also some primary malignant tumours notably osteosarcoma and Ewing's tumour. Chondrosarcoma and fibrosarcoma typically occur in older people (40-60 years). Myeloma, the commonest of all primary malignant bone tumours, is seldom seen before the age of 60 years. In patients above 70 years of age, metastases are more common than all primary tumours together.
- Patients may be completely asymptomatic and the lesion is discovered accidentally on X-ray or as a result of pathological fracture.
- Symptomatic cases mostly will have pain, swelling and local tenderness.
- Differentiation between benign and malignant bone lesions is not always easy, but rapid growth, warmth, tenderness and ill-defined edges suggest malignancy.

Investigations

Plain X-ray should always be the first line of investigation and may even suggest the histological type of the tumour. The site of the tumour may suggest the diagnosis as shown in Table 54.2.

Lesions that simulate primary bone tumours on radiography

1. Secondary tumours.

2. Osteomyelitis.
3. Callus from unnoticed stress fracture.
4. Simple bone cyst.
5. Aneurysmal small bone cyst.
6. Brown tumour of hyperparathyroidism.
7. Fibrous dysplasia.
8. Myositis ossificans.

CT scan and/or MRI.

Assess the site, size and extension of the lesion. They can assess the extraosseous extension of the lesion.

Bone isotope scan is particularly useful for detection of metastases.

Biopsy should be carefully planned. Unless the tumour site is inaccessible, open biopsy should be performed by the surgeon who is to perform the definitive surgery. The biopsy site should be planned after consulting the radiologist and pathologist and is to be done while a tourniquet is applied to the limb to minimize the risk of tumour dissemination. The incision should be longitudinal, through a muscle belly to avoid tumour spread through fascial planes. The edge of the tumour is the ideal part to biopsy because:

- The center may be necrotic.
- To obtain tumour and normal surrounding tissue to determine the microscopic extent of the tumour spread.

Table 54.2. Classification of bone tumours according to their site

Vertebrae	Flat bones	Long bones
Osteoid osteoma	Secondaries	**Epiphysis**
Osteoblastoma	Myeloma	Giant cell tumour
Haemangioma	Chondrosarcoma	Chondrosarcoma
Myeloma		**Metaphysis**
Secondaries		Osteosarcoma
		Diaphysis
		Secondaries
		Ewing's sarcoma

TNM staging

This system can be applied to all primary bone tumours except multiple myelma, juxtacortical chondrosarcoma and parosteal osteosarcoma.

T_X Primary tumour cannot be assessed.
T_0 No evidence of primary tumour.
T_1 Tumour confined within the cortex.
T_2 Tumour invades beyond the cortex.

N Regional lymph node cannot be assessed.
N_0 No regional lymph node metastasis.
N_1 Regional lymph node metastasis.

M_0 No distant spread.
M_1 Evidence of distant metastasis.

Treatment

Treatment of bone tumours calls for close cooperation and consultation between the orthopaedic surgeon, radiologist, pathologist and, certainly in the case of malignant tumours, the oncologist, prosthetic designer and rehabilitation therapist as well.

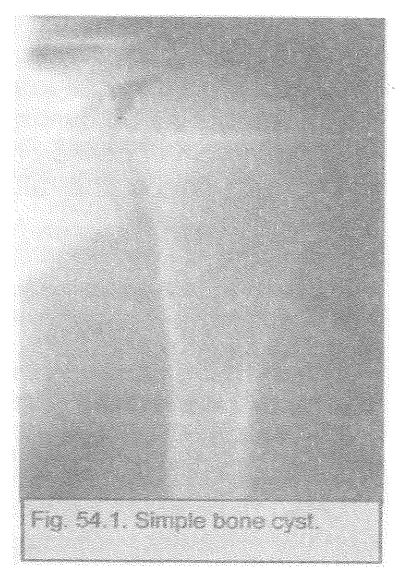
Surgery

1. **Amputation**, if possible, is still the main line of treatment for many primary malignant bone tumours.
2. **Limb preserving surgery**. Successful limb salvage procedures consist of tumour resection, skeletal reconstruction and soft tissue and muscle transfer. The surgical guidelines and techniques of limb- sparing surgery are summarized as follows:
    - No major neurovascular tumour involvement.
    - Wide resection of the affected bone with a normal muscle cuff in all directions.
    - En bloc removal of all previous biopsy sites and all potentially contaminated tissue.
    - Resection of the adjacent joint and capsule.
    - Adequate motor reconstruction, accomplished by regional muscle transfer.
    - Adequate soft tissue coverage.

    Osteoarticular defects are most often reconstructed by segmental, custom prosthesis that are fixed to the remaining intramedullary bone. Within the past few years, there has been interest in applying a porous coating to the prosthesis to obtain biologic ingrowth, in the hope of obtaining long- term, perhaps permanent fixation. In addition Titanium devices have been introduced.

Radiotherapy can be used as an alternative to amputation to destroy radiosensitive tumours as in Ewing's sarcoma. t can also be used in combination with chemotherapy as adjuvant therapy and for inoperable lesions because of the size, local spread, metastatic deposits and marrow cell tumours as myeloma and malignant lymphoma.

Chemotherapy. This is now the preferred adjuvant treatment with promising results as neoadjuvant therapy. Drugs currently in use are Methotrexate, Doxorubicin (Adriamycin), Cyclophosphamide, Vincristine and Cis-platinum. The use of adjuvant chemotherapy has dramatically increased overall survival.


Fig. 54.1. Simple bone cyst.

Non-neoplastic tumour-like lesions

Bone cysts

1. Simple bone cyst (solitary cyst).
2. Fibrous dysplasia.
3. Cysts associated with parathyroid osteodystrophy.
4. Hydatid cyst.
5. Aneurysmal bone cyst.

Solitary simple bone cyst

- Solitary bone cyst is a unilocular cavity filled with clear fluid.
- The lesion is most frequently located in the metaphysis of the upper end of the humerus, femur or tibia, but other bones may be affected.
- This is not strictly a tumour, it tends to heal spontaneously and is seldom seen in adults.

- The condition is usually discovered after a pathological fracture or as an incidental finding on X-ray (Fig. 54.1), which shows a well demarcated radiolucent area in the metaphysis with thinning out of cortex and bone expansion.
- Injection of corticosteroids into the cyst (80-160 mg of methyl prednisolone) may lead to its obliteration. If not, it can be evacuated, the wall scraped and the cavity filled with bone chips.

Aneurysmal bone cyst (Fig. 54.2)

- This is a benign tumour-like lesion that may be encountered at any age and in almost any bone; though more often in young adults and in the long bone metaphysis.
- It contains cavities filled with blood and separated by connective tissue septa containing trabeculae of bone or osteoid tissue and osteoclast giant cells.
- Malignant transformation does not occur.
- X-ray shows a well-defined area, often trabeculated and eccentrically placed. Radiologically, it may resemble a giant-cell tumour, but that tumour extends to the articular surface whereas aneurysmal bone cysts are confined to the metaphyseal side of the growth plate.
- This condition is treated by curettage and packing with bone chips.

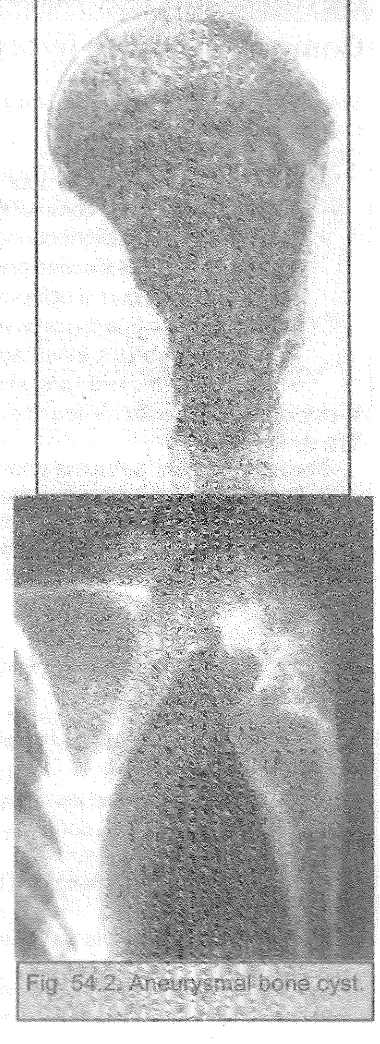


Fig. 54.2. Aneurysmal bone cyst.

Non ossifying fibroma (fibrous cortical defect)

- This is the commonest benign lesion of bone.
- It is a developmental defect in which a nest of fibrous tissue appears within the bone and persists for some years before ossifying.
- It is not a cyst, but it looks like one on X-ray (filled with radiolucent fibrous tissue).
- Clinically, it is asymptomatic and is almost always encountered in children as incidental finding on X-ray; it appears as an oval radiolucent area surrounded by a thin margin of dense bone. It is adjacent to or within the cortex, hence the alternative name fibrous cortical defect is used.
- Most of these defects heal spontaneously, but some may grow, so pathological fracture, is a possibility. Unless fracture occurs, they require no treatment.

Benign bone tumours

Compact osteoma (ivory osteoma)

This is a rare benign tumour, which arises from bones that develop from membrane. It is almost entirely restricted to the skull.

Clinical features

- An adolescent or young adult presents with a painless hard lump, usually on the outer surface of the skull. The tumour is commonly hemispherical (Fig. 54.3)
- Compression on neighbouring structures depends on the position of the osteoma:
  - Growth into air sinuses predisposes to infection and obstruction.

- Deafness occurs if osteoma extends into the acoustic meatus.
- It may grow into the orbit producing distortion of the eyeball.
- If it occurs on the inner table of the skull it, may cause focal epilepsy.
- Stretch of the overlying skin may result in alopecia or adventitious bursa formation.
- X-ray reveals a sessile plaque of exceedingly dense bone with a well-circumscribed edge.

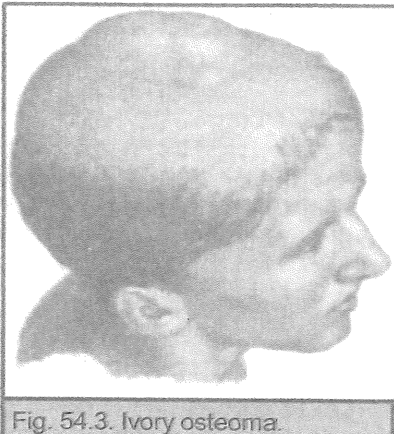


Fig. 54.3. Ivory osteoma.

Treatment

The tumour never turns malignant. Unless it impinges on important structures, it needs not be removed. In the skull an ivory osteoma should never be chiselled off, as this would result in brain concussion.

- If small it may be removed with a trephine.
- If large it is removed with a piece of surrounding bone using Gigli's saw.

Osteoid osteoma

This is a painful benign osteoblastic lesion that is characterized by its small **size** (usually less than one cm, Fig. 54.4).

Patients are usually under 30 years of age and males predominate. Any bone except the skull may be affected, but in over half of the cases it occurs in the femur or tibia.

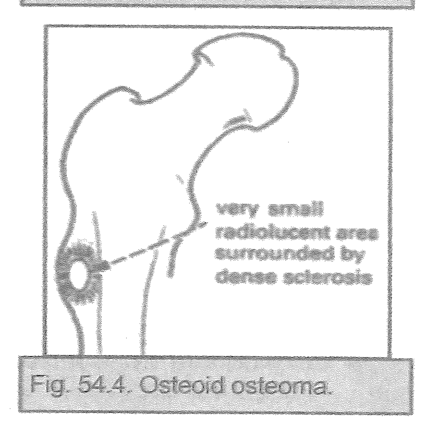


Fig. 54.4. Osteoid osteoma.

Pathology

- Grossly the excised lesion appears as a dark brown or reddish nucleus surrounded by dense bone.
- Microscopically it consists of osteoid tissue with trabeculae of newly formed bone, in a vascular connective tissue matrix.
- There is no risk of malignant transforation.

Clinical features

The leading symptom is pain, which is sometimes severe and is usually relieved by salicylates but not by rest.

X-ray. The important feature is a small radiolucent area, the so-called nidus, which is surrounded by dense sclerosis and cortical thickening. Bone scan shows markedly increased activity.

Treatment is surgical.

The only effective treatment is complete removal of the nidus. The lesion is carefully localized by multiple X-rays and is excised in a small block of bone. The excised specimen should be X-rayed to confirm that it does contain the little tumour.

Chondroma

Chondroma is a relatively common tumour.

Pathology

- Chondroma is a benign tumour that is characterized by the formation of mature cartilage.
- Grossly chondromas are seen in any bone that is preformed in cartilage (most commonly the tubular bones of the hands and feet and less commonly the ribs or the major long bones). Lesions may be solitary or multiple (Oilier's disease). A chondroma is usually situated centrally (enchondroma) or as an eccentric growth (ecchondroma).
- Histologically the appearance is that of simple hyaline cartilage.
- The main complication is pathological fracture (Fig. 54.5).

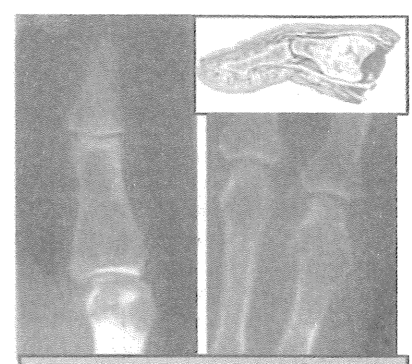


Fig. 54.5. Enchondroma. The right side radiogram shows a pathological fracture.

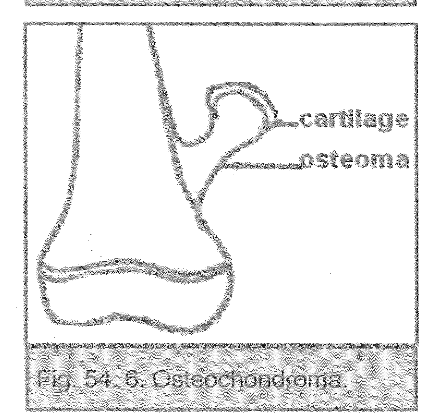


Fig. 54. 6. Osteochondroma.

Clinical features

- Chondromas are seen at any age, but mostly in young people.
- The tumour is usually asymptomatic.
- The patient may present with pain or a swelling or with a fracture after a trivial injury.
- The short long bones of the hands and feet are the commonly affected bones. The lesion may be solitary or multiple.

X-ray shows a well-defined rare area in the medulla, often with characteristic specks of calcification. The solitary-chondroma must be differentiated from a benign cyst (which shows no calcification).

Treatment is not always necessary unless it is large, presents as a pathological fracture or malignancy is suspected. The lesion is excised or curetted and is replaced with bone graft.

Osteochondroma (cartilage-capped exostosis)

This is one of the commonest tumours of bone.

Pathology

- An osteochondroma usually starts in adolescence, as a cartilaginous overgrowth at the edge of the epiphyseal plate and develops by endochondral ossification into a bony protuberance still covered by the cap of cartilage (Fig. 54.6).
- The tumour continue to grow as long as the parent bone grows; any further growth after that is suggestive of malignant transformation.

Clinical features

- The usual site is the metaphysis of a long bone.
- Osteochondroma may be single or multiple (hereditary multiple exostoses, chapter53).
- The patient is usually a teenager or young adult when a painless lump is first discovered.
- Occasionally there is pain due to an overlying bursa or impingement on soft tissue, or rarely, parathesia due to stretching of an adjacent nerve.

- Features suggestive of malignancy include pain, rapid growth, invasion of the mother bone, and recurrence after excision.

X-ray (Fig. 54.7). The radiological appearance is pathognomonic. There is a well-defined exostoses emerging from the metaphysis, its base is continuous with the parent bone. It looks smaller than it feels because the cartilage cap is usually invisible.

Treatment. If the tumour causes symptoms, it should be excised at its base.

Fig. 54.7. Osteochondroma.

Giant cell tumour (osteoclastoma)

Giant cell tumours are discussed separately because they may present with different grades of aggression, from totally benign to frankly malignant:

Pathology

Origin

Giant cell tumour is a lesion of uncertain origin, that derives its name from multinucleated giant cells, which are uniformly distributed in the tumour tissue. It seems to occur only in mature bone i.e., those in which the epiphyses have fused.

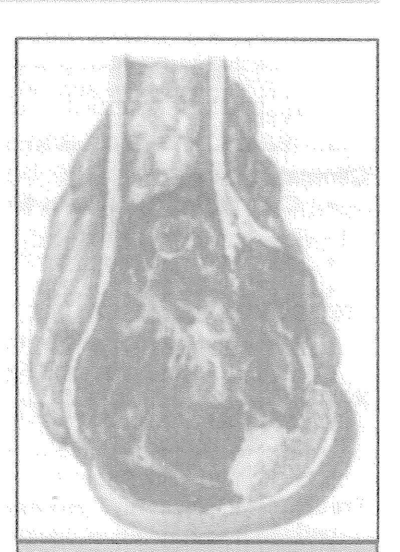


Fig. 54.8. Gross appearance of giant cell tumour.

Gross picture (Fig. 54.8)

- Common sites are the distal femur, proximal tibia, proximal humerus and distal radius, but other bones may be affected.
- It characteristically extends up to the subarticular bone, but usually does not invade the articular cartilage.
- The tumour has a reddish brown, fleshy appearance; it comes away in pieces quite easily when curetted, but it is difficult to remove completely from the surrounding bone.

Microscopic picture (Fig. 54.9)

The striking feature is an abundance of multinucleated giant cells scattered on a background of stromal spindle shaped or ovoid cells, which may be responsible for determining the aggressiveness of the tumour. The numerical grading system (I, II and III) has now largely been discarded as a useful predictor of biological behavior. Accordingly, all giant cell tumours are considered potentially malignant.

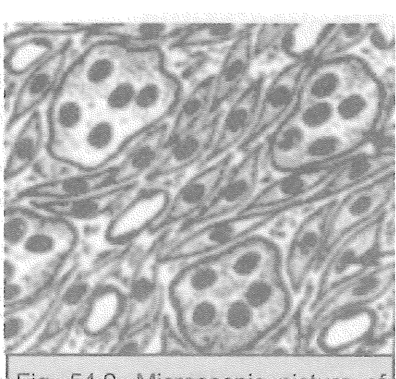


Fig. 54.9. Microscopic picture of giant cell tumour.

Clinical features

Symptoms

- The patient is usually a young adult (20-40 years of age) who complains of pain at the end of a long bone.
- Sometimes there is slight swelling.
- History of trauma and pathological fracture is reported in 10- 15% of cases.

Signs

- There is a vague swelling of the end of a long bone.
- The consistency depends on the degree of thinning of the expanded cortex, it may be soft, firm or an egg-shell crackling sensation may be detected.
- The neighbouring joint is often irritated.

Investigations

X-ray shows a radiolucent area situated eccentrically at the end of long bone and bounded by the subchondral bone plate. It has the characteristic soap bubble appearance due to reactive bone formation under the periosteum. Important is the presence of the medullary plug (operculum) at the junction of the shaft with the tumour. The lack of this plug may signify either malignant osteoclastoma or bone secondaries.

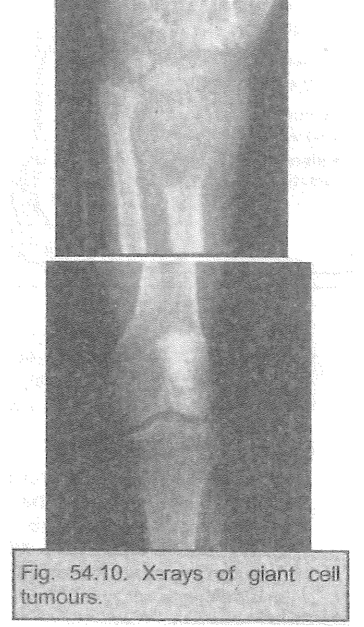


Fig. 54.10. X-rays of giant cell tumours.

Staging. Because of the tumour's potential for aggressive behaviour, detailed staging procedures are essential. CT scans and MRI will reveal the extent of the tumour, both within the bone and beyond. It is important to establish whether the articular surface has been breached; arthroscopy may be helpful.



Treatment

Surgery is the main line of treatment.

- The simplest treatment is curettage and bone grafting, but recurrence is common.
- Wide excision is the treatment of choice, with replacement by specially designed prosthesis or by osteocartilaginous grafts.
- Amputation is indicated for tumours which recur with increasing evidence of malignancy.

Radiotherapy is reserved for surgically inaccessible tumours.

Primary malignant bone tumours

Osteosarcoma

Osteosarcoma is a highly malignant tumour. It is the most common of primary tumours of bone. Osteosarcoma occurs predominantly in children and adolescents. Much less commonly it affects elderly people who have Paget's disease of bone.

Predisposing factors

1. Irradiation.
2. Paget's disease of bone.

Pathology

Origin

The cell of origin is the primitive osteoblasts.

Gross picture (Fig. 54.11)

- The tumour is usually situated in the metaphysis of a long bone. The rule of 80: 80% of osteosarcoma occur in teenagers, 80% in the lower limb, 80% around the knee, 80% in the end of the femur, 80% in the metaphysis.

- Osteosarcoma destroys and replaces normal bone. It is also characterized by the formation of bone or osteoid tissue.
- The tumour rapidly infiltrates the medulla towards the shaft, but it respects the epiphyseal cartilage and hence does not invade the epiphysis or the joint.
- There are four main pathological features.
    - Bone destruction.
    - Tumour bone formation (radiologically appears as sunray appearance).
    - Reactive bone formation (radiologically appears as Codman's triangle).
    - Soft tissue infiltration.
- The tumour varies greatly in appearance. It may be soft, fleshy and vascular with areas of haemorrhage and necrosis (osteolytic type), or it may be solid and contains bone (sclerosing type).

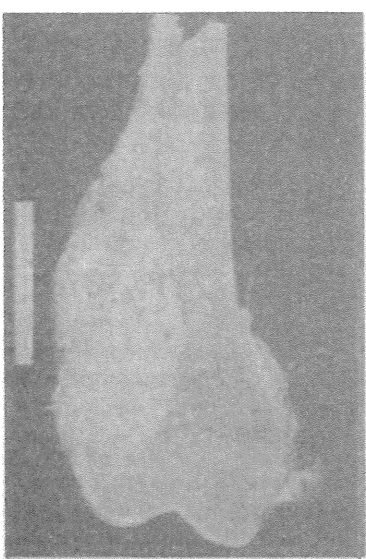


Fig. 54.11. Gross picture of osteosarcoma. Notice:
- The tumour arises in the metaphysis of a child.
- Commonest site is lower femur.
- Tumour invades towards the shaft and outwards into soft tissue but not the epiphysis.

Microscopic picture

The histological appearances show considerable variation. Some areas may have the characteristic spindle cells with a pink-staining osteoid matrix; others may contain cartilage cells or fibroblastic tissue with little or no osteoid. Pathologists are reluctant to commit themselves to the diagnosis unless they see evidence of osteoid formation.

Clinical features

Symptoms

- The incidence of Osteosarcoma is highest between the age of 10 and 20 years, but a second peak occurs after 50 years of age due to malignant changes in Paget's disease.
- Pain is usually the first symptom. Pain is constant, worst at night and gradually increases in severity.
- Sometimes, the patient presents with a mass.
- Pathological fracture is rare because the patient is bed ridden because of the pain.

Signs

- There may be little to find except local tenderness.
- In later cases there is a palpable mass and the overlying tissues may appear swollen and inflamed.
- The regional lymph nodes may be enlarged.

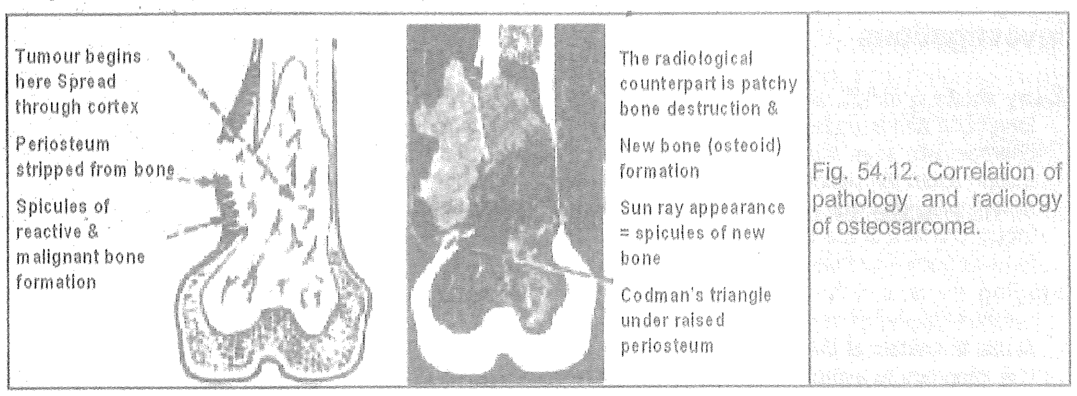
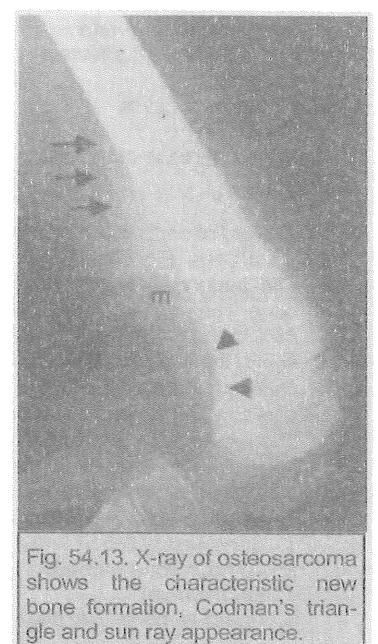


Fig. 54.12. Correlation of pathology and radiology of osteosarcoma.

Investigations

- **X-ray** (Fig. 54.12 & Fig. 54.13) shows hazy osteolytic areas, which may alternate with unusually dense osteoblastic areas. Often the cortex is breached and the tumour extends into the adjacent tissue; when this happens, streaks of new bone formation appear, radiating outwards from the cortex; the so called sunburst (sun-ray) effect. Where the tumour emerges from the cortex, reactive new bone forms at the angles of periosteal elevation (Cbdman's triangle). Both sunray appearance and Codman's triangle are typical of osteosarcoma, yet they may occasionally be seen in other rapidly growing tumours.
- CT and MRI (Fig. 54.14) reliably show the extent of the tumour.
- Chest X-ray may show pulmonary metastasis.
- Biopsy is essential to establish the diagnosis.
- Laboratory tests. The ESR is usually raised and there may be increase in the serum alkaline phosphatase level.


Fig. 54.13. X-ray of osteosarcoma shows the characteristic new bone formation, Codman's triangle and sun ray appearance.

Differential diagnosis

1. Chronic non-specific osteomyelitis.
2. Giant cell tumour.
3. Other malignant bone tumours as chondrosarcoma, fibrosarcoma, Ewing's sarcoma and reticulum cell sarcoma.
4. Secondary carcinomatous deposits.

Treatment

1. Local control of the disease is by either amputation or wide local excision and prosthetic replacement. The level of amputation should be proximal to the joint above the tumour, e.g., osteosarcoma at the tibia is treated by an above knee amputation.
2. Adjuvant chemotherapy has markedly improved the prognosis.


Fig. 54.14. MRI showing osteosarcoma of lower femoral metaphysic in a child with soft tissue invasion.

Chondrosarcoma

- Chondroarcoma is a malignant tumour that is characterized by the formation of cartilage.
- It can occur either as a primary tumour (primary chondrosarcoma) or as a secondary change in a pre-existing cartilaginous lesion most frequently in metaphyseal aclasis or multiple enchondromatosis (secondary chondrosarcoma).
- Both types have their highest incidence in the fourth and fifth decades and men are affected more often than women.
- The tumour is slowly growing and patients may complain of a dull ache or a gradually enlarging lump. Medullary lesions may present as a pathological fracture.

Primary chondrosarcoma is a rare tumour, that may occur in any bone that develops in cartilage but it commonly arises in the pelvic bones, ribs or in the metaphysis of one of the tubular bones. It starts as a central (medullary)

tumour similar to chondroma and expands slowly. This form is seldom seen below the age of 40. Xray shows a destructive medullary tumour containing characteristic flecks of calcification (Fig. 54.15).



Fig. 54.15. Primary chondrosarcomas of the left hip bone and a rib. Both show the characteristic flecks of calcification.

Secondary chondrosarcoma usually arises in the cartilage cap of an exostosis. This malignant change of osteochondroma is suspected by the following:

1. Progressive enlargement of osteochondroma after the end of normal bone growth.
2. Rapid increase in size.
3. Onset of pain.
4. Recurrence after local excision.

Treatment

Surgery. Chondrosarcoma tends to metastasize late, and at least one attempt at wide local excision is justified. Where neither excision nor amputation is feasible, radiotherapy may be used but the response is poor.

Fibrosarcoma of bone

Pathology

- Fibrosarcoma is rare in bone and is more likely to arise in previously abnormal tissue (a bone infarct, fibrous dysplasia or after irradiation).
- The tumour is slowly growing.
- Histologically the lesion consists of masses of fibroblastic tissue with scattered atypia and mitotic cells.

Clinical features

- Fibrosarcoma occurs in older patients than do osteosarcoma (20-60 years of age).
- The patient complains of pain which is less severe than that of osteosarcoma, swelling and there may be a pathological fracture.

X-ray shows an undistinctive area of bone destruction.

Treatment

- Low grade, well-confined tumours can be treated by wide excision with local prosthetic replacement.
- High grade lesions require radical excision or amputation. If this cannot be achieved, local excision must be combined with radiation therapy.

Ewing's sarcoma

Pathology

- This is a rare malignant tumour that arises from vascular endothelium in the bone marrow.
- It usually occurs in the diaphysis of a long bone and gives rise to a periosteal reaction.

Clinical features

- The tumour occurs most commonly between the ages of 10 and 20 years.
- It commonly affects tubular bones, especially in the tibia, fibula or clavicle.
- Pain and swelling are the chief presenting features.
- The swelling is warm and tender, ill-defined and is diaphyseal.

- There may be intermittent or continuous pyrexia, elevated ESR and high leucocytosis. The clinical features may resemble those of osteomyelitis.
- Blood-born metastasis may occur to other bones and to the lungs. Lymphatic spread to regional lymph nodes is common.

Investigations

- **X-ray** usually shows an area of bone destruction which is predominantly diaphyseal; with new bone formation in layers along the shaft (onion peel effect). More often the tumour extends into surrounding soft tissues and may show sun-ray appearance and Codman's triangle.
- **Bone scan** may show multiple areas of activity in the skeleton.
- **CT and MRI** reveal the large extra-osseous component of the tumour and its local extent.

Differential diagnosis

1. Secondaries from neuroblastoma. Usually the patient is below 5 years of age.
2. Reticulum cell sarcoma. Usually occurs in patients above 20 years of age.
3. Osteomyelitis. Biopsy will establish the diagnosis.

Treatment

Treatment of Ewing's sarcoma is somewhat controversial. The prognosis is always poor despite the fact that the tumour is radiosensitive. The best results are achieved by combination of chemotherapy, surgery and radiotherapy; then a further course of chemotherapy for one year is given.

Multiple myelomas

Pathology

Origin

Myelomas are believed to arise from plasma cells of the bone marrow.

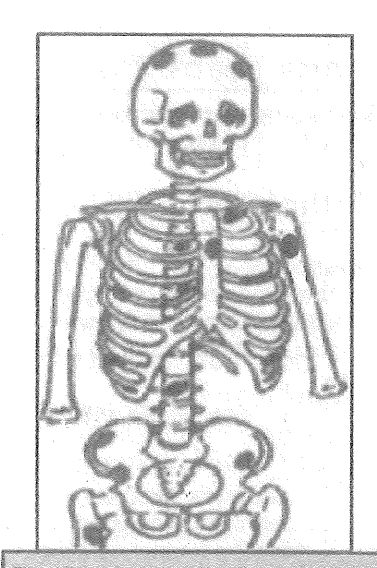


Fig. 54.16. The distribution of red bone marrow in adults. These are the sites of affection with:
1. Metastases.
2. Multiple myeloma.
3. Reticulum cell sarcoma.

Gross picture

- Myelomas are found wherever red marrow occurs; that is, in the trunk bones, the skull and the proximal ends of femur and humerus (Fig. 54.16).

Microscopic picture

The typical microscopic picture is of sheets of plasma cells. A plasma cell has a large eccentric nucleus that contains a spike- like arrangement of chromatin.

Complications

1. Visceral involvement (liver, spleen and lymph nodes).
2. Myeloma kidney (blocking of tubules with protein casts).
3. Metastatic calcifications.
4. Amyloid disease.

Clinical features

- The common age of affection is between 45-65 years.

- Patients present with weakness, bone pain or a pathological fracture. The bone pain is constant and backache is particularly common, sometimes with root pain and occasionally paraplegia.
- Anaemia, cachexia and chronic nephritis contribute to the general ill-health.

Investigations

X-ray. There is overall reduction of bone density; myeloma is one of the causes of secondary osteoporosis. Sometimes there are multiple punched out defects with no marginal new bone round them (similar to metastatic bone lesions, Fig. 54.17).

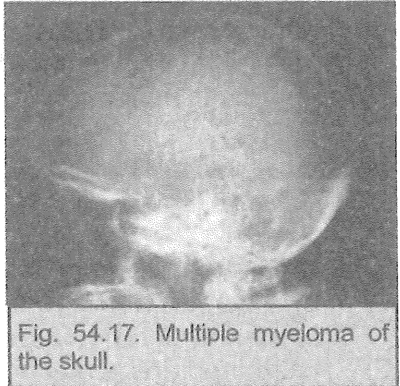
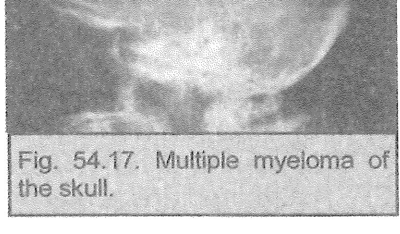



Fig. 54.17. Multiple myeloma of the skull.

Laboratory findings

1. Urine analysis reveals Bence Jones protein (coagulates at 55°C and disappears at 85°C).
2. Plasma and urinary protein electrophoresis shows a characteristic pattern.
3. Sternal puncture reveals the typical myeloma cells (plasma cells form 10% or more of the nucleated cells of the marrow).
4. Blood analysis reveals anaemia, raised ESR (osteoporosis + a high ESR= myelomatosis until proved otherwise), raised alkaline phosphatase, elevated gamma globulin, hypercalcaemia and sometimes plasma cell leukaemia (where myeloma cells enter the circulation and elevate the white cell count).

Differential diagnosis

1. Bone secondaries.
2. Parathyroid osteodystrophy.
3. Osteoporosis.

Treatment

- Radiotherapy and chemotherapy relieve pain and pressure effects for a time, and may prolong survival.
- Surgery has a limited role for complicated cases. Pathological fractures in the limbs are best treated by internal fixation. Unrelieved cord pressure may need decompression.

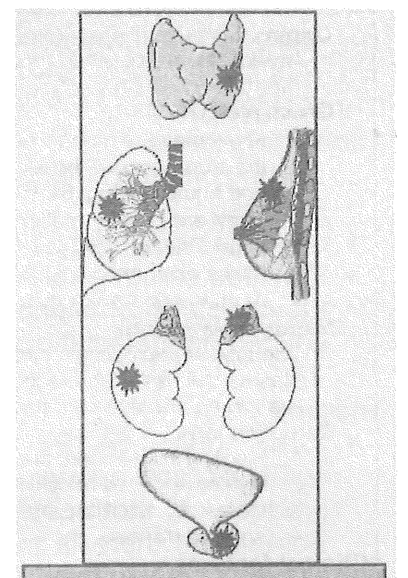


Fig. 54.18. Sites of primary malignancies that commonly metastasize to bones.

Bone metastases

Metastatic deposits are seen more frequently than all primary bone tumours together.

Pathology

Sources

The commonest sources are carcinoma of the breast, prostate, kidney, lung, thyroid, and adrenal neuroblastoma (Fig. 5418). In about 10% of cases no primary tumour is found.

Site

Metastases usually appear in areas containing red marrow (Fig. 54.16).

Types

1. Osteolytic deposits. These destroy and replace bone, partly by their own expansion and partly by stimulating active bone resorption (osteolytic lesions) by direct action of the tumour cells or tumour derived factors that stimulate osteoclastic activity.
2. Osteoblastic (bone-forming) deposits are uncommon. They usually occur in prostatic carcinoma.

Clinical features

- Patients are usually aged 50-70 years.
- Pain is the commonest and often the only clinical feature.
- Sometimes nothing is suspected until a pathological fracture occurs.
- The primary tumour may be obvious, but sometimes even a meticulous search fails to reveal it. The neck, breasts, axillae, lungs, abdomen and genitalia should be examined and rectal or vaginal examination is usually necessary.
- Symptoms of hypercalcaemia may occur (often missed). These include anorexia, nausea, abdominal pain, general weakness, depression and polyuria.

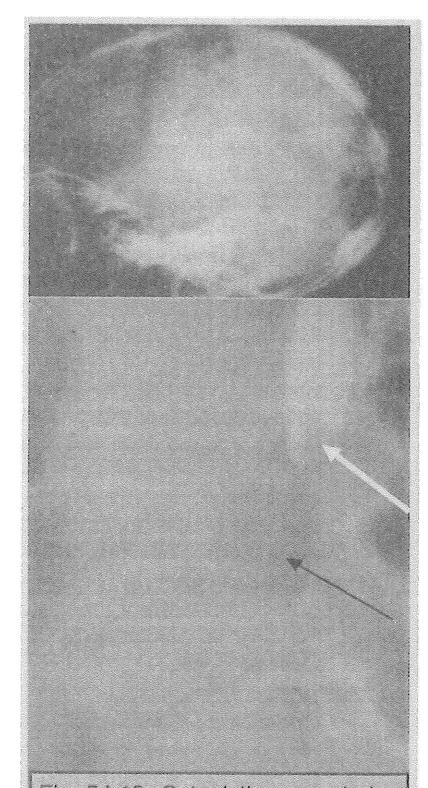


Fig. 54.19. Osteolytic secondaries of the skull and the spine. The latter (black arrow) compresses the spinal cord as shown by interruption of myodil (contrast in subarachnoid space) as pointed by the white arrow.

Investigations

Laboratory tests

The common findings are:

- Elevated ESR.
- Low haemoglobin.
- Elevated serum alkaline phosphatase.
- Elevated acid phosphatase and PSA (prostate specific antigen) in metastatic prostate carcinoma.

Imaging

- X-ray (Fig. 54.19). Most skeletal deposits are osteolytic and appear as rare areas in the medulla or produce a moth-eaten appearance in the cortex. Sometimes there is marked bone destruction with or without pathological fracture. Osteoblastic deposits usually signify prostate carcinoma.
- CT scan (Fig. 54.20)
- Radioscintigraphy with ^{99m}TcHDP is the most sensitive method of detecting silent metastatic deposits in bone (Fig. 54.21). Areas of increased activity are selected for X-ray examination.

Treatment

Metastatic carcinomas usually have a bad prognosis; yet, patients deserve to be made comfortable, to enjoy, as far as possible, their remaining months or years, and to die in a peaceful and dignified way.

Treatment is palliative.

1. Painful deposits are treated by radiotherapy.
2. Pathological fractures are treated by a combination of internal fixation and radiotherapy.
3. Chemotherapy.



Fig. 54.20. CT scan showing two metastatic deposits in a vertebra.

Metastases are more frequent than primary malignancies in the following organs:

1. Lungs
2. Liver
3. Bones
4. Brain

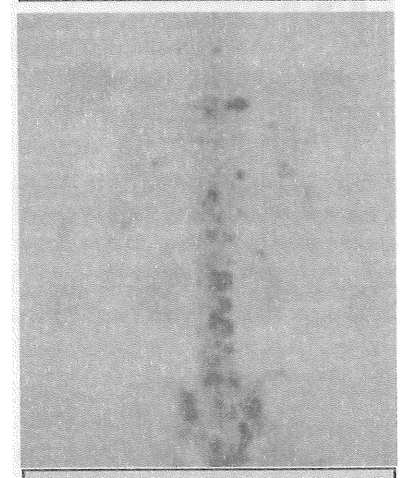


Fig. 54.21. Bone isotope scan showing areas of increased uptake (darker spots) in the vertebrae, ribs and iliac bone. These are suggestive of bone metastases.

DEFORMITIES OF BONES AND JOINTS

Deformities of the elbow

Normally the forearm is in a position of slight abduction with the arm constituting an angle of about 10-15° known as the carrying angle. In cubitus valgus, this angle is increased so that the hand deviates away from the body while in cubitus varus the reverse occurs (Fig. 55.1).

CHAPTER CONTENTS

Cubitus valgus

The most common cause is nonunion of a fractured lateral condyle of the humerus. This may give gross deformity and a bony knob on the inner side of the joint. The importance of valgus deformity is the liability for delayed ulnar palsy to develop. Years after the injury, the patient notices weakness of the hand with numbness and tingling of ulnar distribution. The deformity itself needs no treatment but for delayed ulnar palsy, the nerve should be transported to the front of the elbow.

Cubitus varus

The most common cause is malunion of a supracondylar fracture of the humerus. The deformity is obvious when the elbow is extended and the arms are elevated; it looks ugly and the hand brushes against the body in walking. The deformity can be corrected by a wedge osteotomy of the lower humerus.

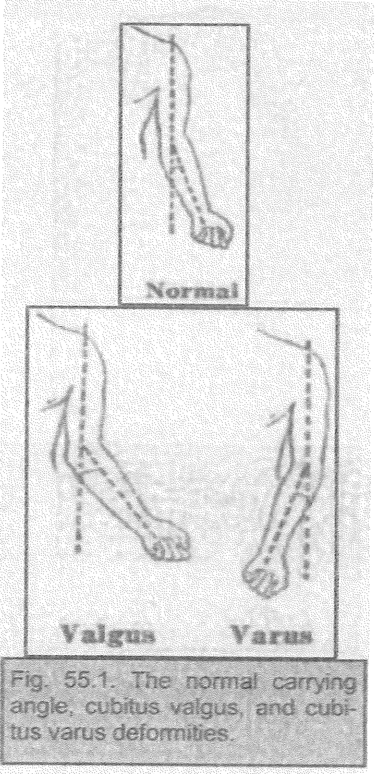


Fig. 55.1. The normal carrying angle, cubitus valgus, and cubitus varus deformities.

Deformities of the hip

Congenital dislocation (developmental dysplasia of the hip)

In this disorder of newborn children, the hip is dislocated either at birth or soon afterwards. Unless corrected it leads to permanent deformity and disability.

Incidence

The incidence is about 1 in 1000 newborn babies. It is much more common in girls than in boys. The left hip is more often affected than the right. In nearly a third of all cases, both hips are affected.

Aetiology

1. There is a familial tendency.
2. Intrauterine malposition has been blamed. These babies have a higher than usual incidence of breech presentation.

Pathology

- The acetabulum is unusually shallow.
- The femoral head slides out posteriorly or laterally and then rides upwards.
- The joint capsule though stretched remains intact and by folding inwards may impede reduction.
- The fibrocartilagenous labrum is often turned into the acetabulum and this acts as a further obstacle to reduction,
- Maturation of the acetabulum and femoral epiphysis is retarded and the femoral neck is short and anteverted.
- Neglected cases may suffer hip osteoarthritis in later years.

Normal Diseased

Fig. (55.2): Trendlenburg's test

Clinical features

Mother's observations

- Before the baby starts walking, an observant mother may spot asymmetry, a clicking hip or difficulty in applying the napkins because of limited abduction.
- After walking starts, asymmetry is more obvious and now a limp also becomes apparent.

Congenital hip dislocation should be diagnosed soon after birth where treatment is simple and most effective.

Examination

- With unilateral dislocation, the skin creases lie at different levels on the two sides. The affected leg is slightly short and rotated externally. Trendelenburg's test is always positive (Fig. 55.2). Standing on one foot, on the side of the dislocated hip, the pelvis drops on the unsupported (healthy) side. This is caused by impairment of abductor muscles on the dislocated side.
- Bilateral dislocation is more difficult to detect b&ause there is no asymmetry and the characteristic waddling gait may be mistaken for normal toddling, however, the perineal gap is abnormally wide and abduction may be limited.

Special tests

Congenital dislocation should be diagnosed soon after birth where treatment is simple and most effective. Routine postnatal examination must include special tests for instability (Fig. 55.3).

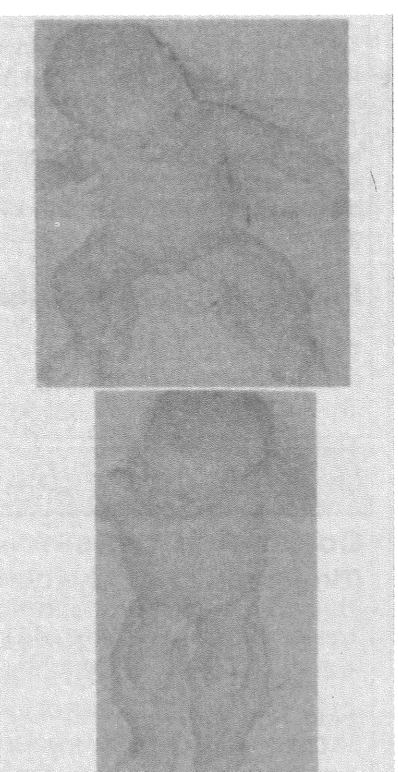


Fig. (55.3): Special tests. *Upper* Oroalani's test. *Lower* Barlow's test

1. **Ortolani's test**. The baby's thigh is held with the middle finger behind the greater trochanter and the thumb in front; during abduction the greater trochanter is pressed forwards and during adduction the femoral head is levered gently backwards. The test is positive if there is a click or a jerk during the abduction manoeuvre indicating that the hip has been reduced.
2. **Barlow's test**. If the hip is already reduced, the adduction manoeuvre will allow the femoral head to

be gently levered out of the socket (Barlow's modification of Ortolani's sign). Unfortunately these tests are sometimes inconclusive and a mild instability may still b missed only to reappear as a full blown dislocation 3 months later.

Investigations

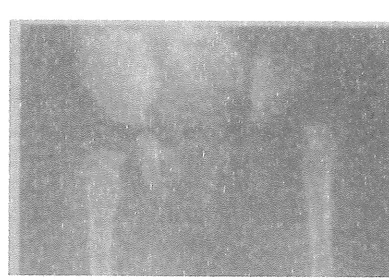


Fig. (55.4): Congenital dislocatior of left hip

- X-ray examination is of little value in the newborn because the head of the femur is entirely cartilagenous and not visible radiologically. In a child of one year age or more it shows:
  - The acetabular socket is unusually shallow.
  - The ossific centre of the femoral head is underdeveloped and is displaced upwards and outwards (Fig. 55.4).
  - Ultrasound scanning can visualize the shape of the acetabulum and the position of the femoral head with reasonable accuracy. It is particularly useful in the newborn, when radiography is undependable.

Treatment

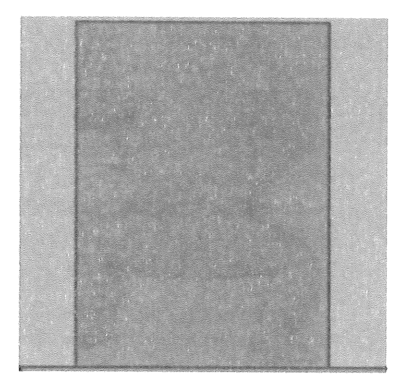


Fig. (55.5): vonRosen splint maintains the hips in abduction

In the newborn. Most hips with a positive instability sign at birth become normal without treatment. To avoid unnecessary splinting, some surgeons advocate waiting 3 weeks; if the hip is still unstable, it is reduced and splinted in abduction. However if ultrasound scanning soon after birth shows that the acetabular roof is defective, it is far wiser to start splinting using vonRosen splint (Fig. 55.5). This is maintained for 3-6 months by which time scanning should show a good acetabulε roof.

Cases discovered between the ages of 6 months to 6 years. The objectives ar to reduce the hip and hold it reduced until acetabular development is satisfactory.

- Closed reduction in a plaster cast with the hips abducted. This position is maintaine for 6 weeks. The plaster is then replaced by a splint which prevents adduction bι allows movement. Within 12 months X-ray may show a nicely reduced femoral hea with a normal acetabular roof, if so, the splint is discarded.
- Operation. Cases that fail to reduce are treated by open reduction followed by fixatio in a plaster cast. A markedly shallow acetabulum can be deepened by a concomitar pelvic osteotomy.

Cases discovered after the age of 6 years

- For unilateral dislocation, operative reduction is the preferred treatment even thoug the prognosis is uncertain. As in the former group, it may be necessary to combin this with corrective osteotomy.
- With bilateral dislocation the deformity and waddling gait are symmetrical an therefore not so noticeible. The risk of operative intervention is also greater becaus failure on one or other side turns this into an asymmetrical deformity. Therefore, i these cases most surgeons would avoid surgery unless pain or deformity is severe.

Coxa vara

Coxa vara is a diminution of the neck shaft angle of the femur below the normε 120°-140°.

Deformities of the knee

Genu valgum (Knock knees)

This is a fixed abduction deformity of the knees from the middle line. When the knees are placed in contact, the malleoli separate from each other (Fig. 55.6).

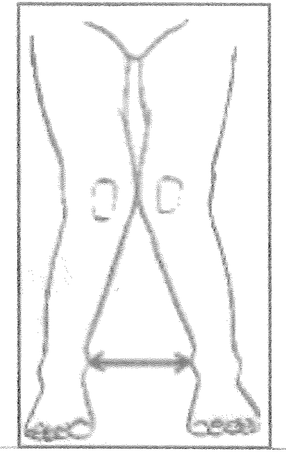


Fig. (55.6): Genu vaglum

Aetiology

1. Idiopathic; this is the commonest. It is invariably bilateral.
2. Bone softening occurring with rickets and rheumatoid arthhtis.
3. Bone injury with epiphyseal damage or following a fractured lateral tibial condyle.

Clinical features

- With idiopathic knock knee, deformity is the only symptom. It appears at the age of 2-3 years and nearly always recovers by the age of 6 years. Other postural deformities such as flat foot may coexist but these children are normal in other aspects.
- In the other varieties, there may also be symptoms and signs of the underlying cause.
- The deformity appears when the patient stands but disappears when the knees are flexed. The knees tend to knock together so that the patient walks with the knees partially flexed and separated and tends to fall during running.

Treatment of the idiopathic type

- The parents should be reassured and the patient is observed as the condition usually requires no treatment.
- If by the age of 3 years the intermalleolar distance is more than 3 inches this is an indication for corrective osteotomy.

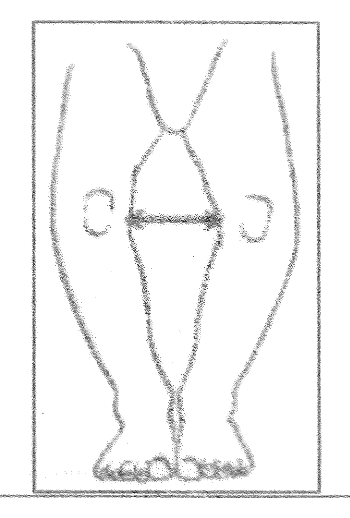


Fig. (55.7): Genu varum

Genu varum (Bow legs)

There is a lateral bowing of the legs so that the knees are widely separated when the malleoli are in contact, and the knees are held straight (Fig. 55.7).

Aetiology

1. **Physiological**. This is the commonest variety, some babies are borne with slight bow leg or develop it while wearing napkins. These children nearly all grow straight.
2. Bone softening due to rickets and Paget's disease.
3. Bone injury affecting the epiphyseal site and sometimes following fracture of the upper tibia.

Clinical features

The bowing may involve the tibia alone or the femur may also be affected. The deformity is ugly and causes the patient to look short and stunted.

Treatment
- Idiopathic bow legs usually recover.
- If the bow legs are severe, operation is desirable both for cosmetic reasons and to prevent osteoarthrosis.
    - Stapling the lateral side of the lower femoral epiphysis.
    - Infra-condylar tibial osteotomy after 5 years.

Deformities of the foot
Congenital talipes equinovarus (Club foot)
Terminology
- Congenital — The deformity is present at birth
- Talipes — Ankle and foot
- .Equino — Plantar-flexion of ankle that looks like a horse's hoof.
- Varus — Inward turning of the foot. The sole looks inwards.

Incidence
This is a relatively common deformity (1 in 1000 live births). The condition is bilateral in one third of cases.

Aetiology
The cause is unknown

Predisposing factors include
1. Positive family history.
2. Male sex. Boys are affected twice as often as girls.

Pathological anatomy
- The deformity includes three anatomical disturbances.
    - Planter flexion of the foot at the ankle.
    - Inversion of the os calcis and of the navicular on the talus.
    - Adduction of the bones of the forefoot at the mid tarsal and taso-metatarsal joints.
- Soft tissues are also affected:
    - Shortening of all ligamentous structures on the medial side of the foot.
    - Hypoplasia of the muscles of the calf leading to shortening of the flexors of the foot and toes.
- If the condition is not corrected early, secondary growth changes occur in bones, these are permanent.

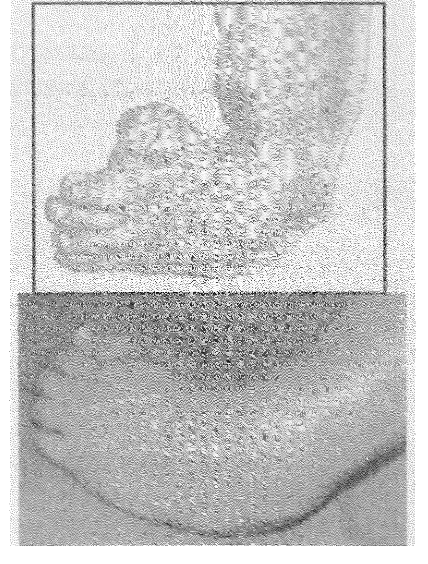


Fig. (55.8): Talipes equinovarus

Clinical features
- Deformity is the only symptom in infancy (Fig. 55.8).
- Painful callosities develop years later in the forefoot if the deformity remains uncorrected.
- The heel may be small and high and the calf is thin,
- Gentle attempts at passive correction show the deformity to be fixed.
- The infant must be examined for associated disorders such as spina bifida or arthrogryphosis.



- X-ray shows the shape and position of tarsal ossific and is helpful in assessing the progress after treatment.

Treatment

Non-operative treatment (manual correction)

Treatment begins within 2 or 3 days of birth. Each component of the deformity is corrected in the following order, first the forefoot adduction, then inversion and finally the equinus (ankle dorsiflexion). Without anaesthesia, the foot is gently manipulated towards the desired position and held there by adhesive strapping or by a plaster cast. The process is repeated weekly for 6-8 weeks until the foot is not only corrected but overcorrected. Finally correction must be confirmed by X-ray.

Surgery

Indications

- Resistant cases
- Older children

Procedure includes

- Reduction by elongation of tendo Achillis, division of tight ligaments.
- Osteotomies are needed for children above 5 years.
- Maintaining reduction by casts then by splints.
- Triple arthrodesis is done for advanced cases.

Flat foot (Pes planus)

At birth the foot is flat and the normal arch is only acquired when the infant stands. The arch is maintained in adult life by the shape of the bones of the foot, their connecting ligaments and the action of the muscles of the calf and sole. Flat foot in adult life may be due to either collapse of normal arches or to the persistence of the infant shape. The following are the varieties of fiat foot.

1. **Congenital** due to congenital vertical talus.
2. **Infantile**. This is physiological.
3. **Spasmodic flat foot**. This is an uncommon painful condition which presents insidiously in adolescence. The foot is everted and hence the medial arch appears flat. The tendons of the peroneal and long toes extensors can be seen standing out in continuous contraction. Attempts to passively invert the foot are resisted by these muscles and are painful. In many patients suffering from this condition, an abnormal bar of bone joining the calcaneus to the talus or to the navicular can be demonstrated. Treatment is by triple arthrodesis.
4. **Idiopathic adult flat foot**. This is the commonest form of symptomatic flat foot in adults. It may be the end result of repeated episodes of foot strain; an ill-defined uncommon condition in which the foot becomes acutely painful following excessive use. More commonly the condition is gradually progressive and presents with aching pain in the foot after long continued standing. It is seen most commonly in people who spend long hours standing. The medial longitudinal arch gradually collapses causing pain cantered in the medial part of the sole. Local tenderness may be found in the sole under the apex of the arch probably because the plantar ligaments are overstretched. Pain is provoked if these ligaments are stressed by dorsiflexing the forefoot. In later stages, the foot becomes painless and the gait shuffling and inelastic. The foot is rigid and flat. All joints of the hind-foot degenerate.

   Treatment is only indicated if the flat foot is symptomatic. The pain of foot strain is relieved by rest. Its prevention may require arch supports. Exercises to improve the function of the intrinsic muscles of the sole and of the toe flexors may prevent further deterioration. When the foot has finally collapsed, little can be done to

improve its appearance or function and the patient should be advised to accommodate his life to his feet by taking a sedentary job.

5. **Traumatic flat foot**. This is caused by fractures which abolish the longitudinal arch. Fractures of the os calcis may produce a severe fiat foot with a rigid subtalar joint.

Hallux valgus

Hallux valgus is the commonest of the foot deformities and probably of all musculoskeletal deformities. It consists of outward deviation of the great toe at the metatarso-phalangeal joint.

Aetiology

1. Hallux valgus is usually due to badly fitting shoes with pointed fronts and high heels.
2. It is rarely due to congenital metatarsus varus in which the first metatarsal is inclined medially away from the other metatarsals.

Pathology

- Outward deviation of the first toe at the metatarso-phalangeal joint (Fig. 16.11).
- The deformity tends to be progressive since the displacement of the flexor and extensor tendons exerts bow string acting which pulls the first phalanx more and more laterally.
- Complications
    - Callosity in the overlying skin
    - Adventitious bursa over the prominent head (bunion)
    - Exostosis over the prominent part of the metatarsal head
    - Osteoarthrosis of the metatarsophalangeal joint.

Clinical features

- Hallux valgus is usually bilateral.
- It is most common in adolescents and in the sixth decade and in females.
- The big toe may lie above or below the second toe and the other toes are pushed laterally and often develop a hammer toe deformity. The head of the first metatarsal projects on the medial side of the foot.
- Features of complications, e.g., pain from a bunion.

Treatment

- **Adolescents**. Deformity is usually the only symptom, but nothing short of operation can prevent the deformity from increasing. This takes the form of a corrective osteotomy of the first metatarsal which once it is straight, the big toe assumes a more normal position.
- **Adults**. All patients can be made more comfortable by careful attention to footwear. The shoes should be wide. Padding may be used to protect the bunion of hammer toe. If the deformity is not too severe, a tendon release of the adductor hallucis on the lateral side of the big toe with trimming of the prominent metatarsal head and tightening of the medial capsule may give adequate correction. If the deformity is severe, excision arthroplasty is indicated. Either the proximal part of the proximal phalanx (Keller's operation) or the first metatarsal head (Mayo's operation) is removed.

AMPUTATIONS

General principles

Indications

Amputation should be considered when part of a limb is Dead Deadly or a Dead loss.

1. **Dead**. The causes of gangrene are outlined in chapter 13.
2. **Deadly**. The life of the patient may be threatened if the limb is retained under the following conditions:
   a. Malignant, tumours including osteosarcoma, soft tissue sarcoma infiltrating the bone and high grade giant cell tumours.
   b. Gas gangrene.
   c. Crush injury that may lead to crush syndrome.
3. **Dead loss**. The affected limb may be considered inferior to an artificial limb or to no limb at all. Amputation in the following circumstances can improve the quality of life.
   a. Severe lacerations and fractures that are not suitable for satisfactory repair.
   b. Severe contracture or paralysis.
   c. Severe rest pain.

CHAPTER CONTENTS

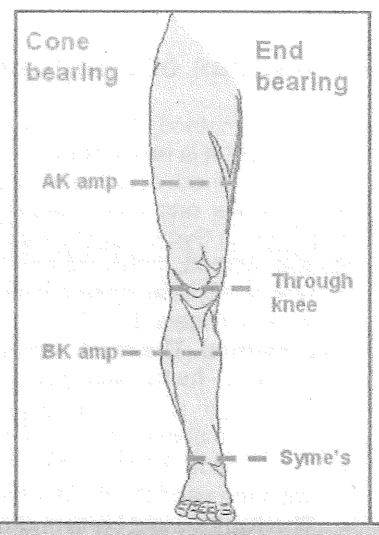


Fig. 56.1. End-bearing and cone-bearing amputations.

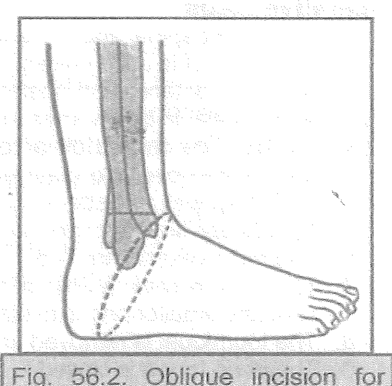


Fig. 56.2. Oblique incision for Syme's amputation.

Types of amputations

1. **Provisional**. This is performed when it is anticipated that primary healing is unlikely to occur because of infection or ischaemia. The amputation is performed at the lowest possible site, so that if further amputation is necessary, it will result in a stump of adequate length. Free drainage should be provided by employing either the guillotine method or better a flap amputation without closure.
2. **Definitive**. When primary healing is most likely to occur, the amputation should be planned to provide an ideal stump. There are two types of definitive amputation (Fig. 56.1):
   a. **End-bearing** amputation is performed when weight is to be taken through the end of a stump. Therefore, the scar must not be terminal and the bone end must be solid not hollow which means it must be cut through or near a joint. Examples are through knee and Syme's amputation.
   b. **Non-end-bearing** (cone-bearing) amputation is the commonest variety. All upper limb and most of lower limb amputations come into this category. In these amputations the body weight is transmitted by way of the artificial limb socket to structures other than the stump end.

Requirements of an ideal amputation stump

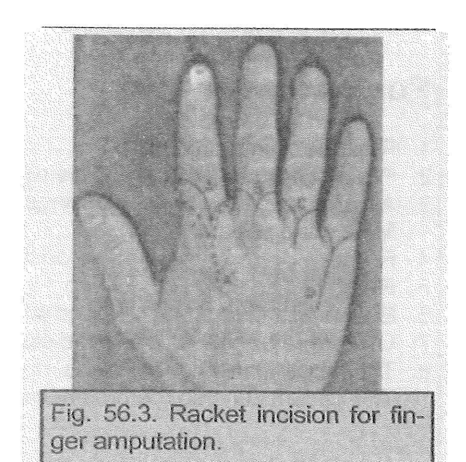
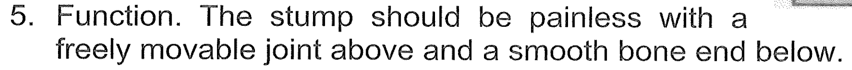


Fig. 56.3. Racket incision for finger amputation.

1. Length. The length of a stump is an advantage because a short stump is liable to slip out of the prosthesis, but a non-end-bearing stump should be 3 inches shorter than the entire bone to give room for the artificial joint.
2. Shape, The stump should be smoothly rounded but neither bulbous nor pointed.
3. Coverings. The bone should be covered with subcutaneous tissues and deep fascia to ensure mobility of the skin.
4. Scar. It should be linear, freely movable and not exposed to pressure. A terminal scar is satisfactory in the upper limb but not in the lower limb when the stump is end bearing.
5. Function. The stump should be painless with a freely movable joint above and a smooth bone end below.



Techniques of amputation

1. **Guillotine method**. All the tissues are divided at the same level and the bone end is left exposed on the cut surface. It is rarely employed nowadays except in extreme emergencies.
2. **Oblique method**. An oblique elliptical incision is made with its upper end above the level of bone section and its lower end lower than this level by a distance equal to the diameter of the limb. This method is employed in Symes amputation (Fig. 56.2).
3. **Racket method**. In this method a straight incision is carried proximally as the handle of the racket from a circular or elliptical incision which represents the blade of the racket. This is employed in amputation of the toes or fingers (Fig. 56.3).
4. **Flap method**. The most popular method is to use flaps which may be a single flap or two equal or unequal flaps. The following rules should be respected in making the flaps:
   a. The length of a single flap or the combined length of the two flaps should be equal to one and half times the diameter of the limb at the level of bone section.
   b. The breadth of equal flaps should be equal to half the circumference of the limb but when unequal flaps are used the shorter flap should be broader than the other.
   c. The flaps should be semicircular rather than rectangular in shape since a conical rather than a cylindrical stump is desired.
   d. The flaps should consist of skin and deep fascia with some muscle tissue at their bases.

Operative steps

1. The consent should be obtained.
2. General or spinal anaesthesia.
3. A tourniquet is used unless there is arterial insufficiency.
4. As a rule anterior and posterior flaps of equal length are used in the upper limb and for above knee amputation. In below knee amputation a long posterior flap is usual.
5. Muscles are divided distal to the proposed site of bone section, subsequently the opposing groups are sutured over the bone end to each other and to the periosteum thus providing a better muscle control as well as better circulation.

6. Nerves are divided proximal to the bone cut.
7. The main vessels are tied.
8. The bone is sawn across at the proposed level after division of the periosteum. If two bones are present, the smaller one is divided at first.
9. The tourniquet is removed and every bleeding point is meticulously ligated.
10. The deep fascia is closed.
11. The skin is sutured without tension. Suction drainage is advised.
12. The stump firmly bandaged.

Postoperative care

1. Rest the stump on a pillow.
2. Prevent flexion deformity of the hip and knee joints by using suitable splints.
3. Avoid change of stump dressings for five to seven days unless excessive oozing occurs or signs of infection are apparent.
4. Remove drain 24 to 48 hours postoperatively.
5. Remove skin sutures between 10 and 14 days.
6. Encourage stump muscles and joint exercise with the help of supervised physiotherapy.
7. Use crepe bandages or elastic stump stockings to achieve a smooth conical stump.
8. The prosthesis. All prostheses must fit comfortably. They should also function well and look presentable. The patient can use the prosthesis once the stump is well healed and conical and the scar is stable.

Complications of amputation

Early complications

1. Complications of any operation especially haematoma, infection and secondary haemorrhage.
2. Special Complications
   a. Breakdown of skin flaps, which may be due to ischaemia or to suturing under excessive tension.
   b. Gas gangrene. Clostridia spores from the perineum may infect a high above the knee amputation especially if performed through ischaemic tissues.

Late complications

1. Skin. Eczema, callosities, ulceration, redundancy, and adherent scar.
2. Muscle. Excess muscles left at the end of the stump gives a sense of insecurity which may prevent the use of proper prosthesis.
3. Artery. Progress of arterial occlusive disease produces ischaemia of the stump.
4. Nerve:
   a. Phantom limb is the conscious feeling of the missing limb which persists for sometime and then fades gradually. The patient may feel pain in his phantom limb.
   b. Stump neuroma is usually symptomless unless irritated by pressure.
   c. Causalgia is a severe burning pain due to the formation of artificial synapses between the efferent sympathetic and the afferent sensory fibres. It is relieved by sympathectomy.
5. Joint. The joint above an amputation may be stiff or deformed.
6. Bone
   a. Formation of spur.
   b. Osteomyelitis and ring sequestrum.
   c. A projecting bone end may result from the use of short flaps or from continued growth of bone in children.

Lower limb amputations

Distal amputations

Local toe amputation. In patients with toe gangrene because of a small-vessel disease the surrounding tissues have relatively good blood supply. Therefore, local amputation of the toe can result in healing.

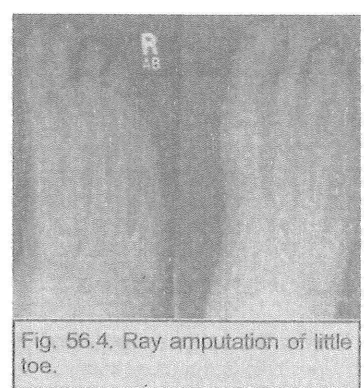


Fig. 56.4. Ray amputation of little toe.

Ray amputation of a toe. When the metatarso-phalangeal joint region is involved, ray amputation is recommended, where a part of the metatarsal bone is excised (Fig. 56.4).

Transmetatarsal amputation is indicated when several toes are affected and irreversible ischaemia has extended to the forefoot. A viable long plantar flap is essential for satisfactory healing of this amputation (Fig. 56.5).

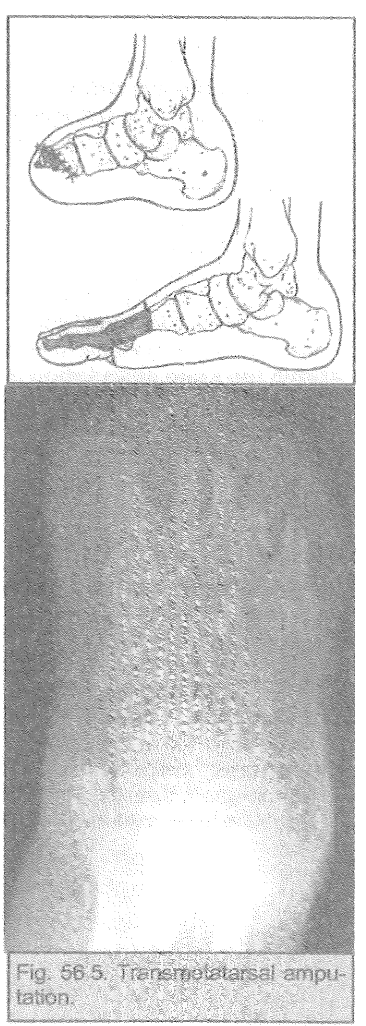


Fig. 56.5. Transmetatarsal amputation.

Syme's amputation is a disarticulation at the ankle with removal of the malleoli and tibial articular surface. It is very satisfactory provided that the circulation of the limb is good.

Advantages. Syme's amputation is better than a below knee amputation because:

- It is less catastrophic to the patient.
- It allows the patient to walk in his room without a prosthesis.
- It maintain the pleasure of earth feeling.
- It requires a cheap stump boot known as elephant boot and not an expensive prosthesis.

Disadvantages

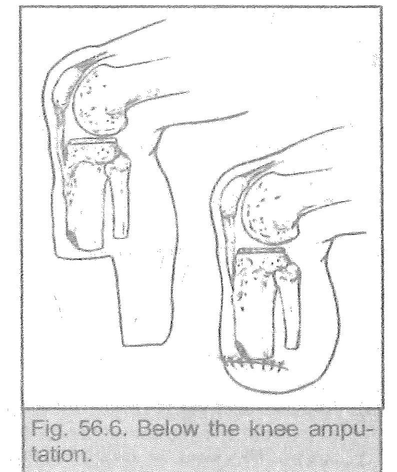
- After 7-10 years reamputation (below the knee) is usually required as the stump is end bearing and results in ulcerations, callosities and exostosis.
- The prosthesis is ugly.

Major amputations

Below the knee amputation (Fig. 56.6)

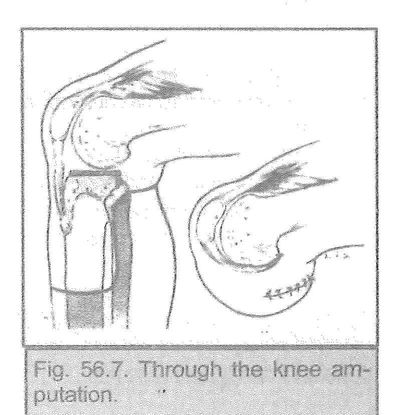
- This is the most common amputation for vascular disease and infection.
- The optimum length is 6 inches below the joint line or one hand breadth below the tibial tubercle because at lower levels the circulation is poor and the bone is not well protected by soft tissues. The stump should not be shorter than 2 inches so that it can be retained inside the socket during flexion of the knee.
- This amputation is done using a long posterior and a short anterior flap because the blood supply of the posterior flap is better than the anterior. The flap should include the deep fascia and where it is deficient over the tibia, the periosteum should be included. The calf muscles are included in the posterior flap.

- The fibula is cut first and to obtain a conical stump it should be divided one inch higher than the tibia. The sharp anterior margin of the tibia should be bevelled.
- Ninety percent of unilateral and 75% of bilateral below knee amputees learn to walk independently.


Fig. 56.6. Below the knee amputation.

Through the knee amputation (Fig. 56.7)

Amputation at the knee is not popular because requirements are seldom available and because the end cannot be fitted with a good artificial limb.

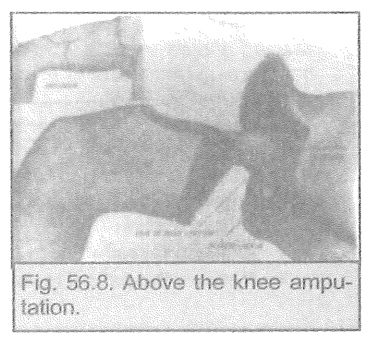

Fig. 56.7. Through the knee amputation.

Above the knee amputation (Fig. 56.8)

- This amputation should be performed when the blood flow is inadequate for healing at a lower level, when the patient is unable to walk because of other debilitating disease or when serious infection precludes lower amputation.
- The chief advantage is the greater likelihood of healing while the chief disadvantage is a lower rate of subsequent ambulation.
- The ideal length is 11 inches from the tip of the greater trochanter and the minimum is 6 inches because at higher levels sufficient adductor power is not retained.
- The amputation can be carried out through equal or unequal anteroposterior flaps.

Hip disarticulation is rarely indicated and very difficult to fit with a prosthesis. The main indication is malignant disease.

Hindquarter amputation (hemipelvectomy) is only performed for malignant disease. The classic operation involves removal of the entire lower extremity and varying amounts of the pelvic bone.


Fig. 56.8. Above the knee amputation.

Upper limb amputations

Distal amputations

Partial finger amputation may be carried out through the phalanx or interphalangeal joints using a single palmar flap.

Complete finger amputation is usually combined with excision of the metacarpal head to minimize the deformity and enable the fingers to be flexed closely. A dorsal racket incision (Fig. 56.3) is employed with the blade encircling the root of the finger and the handle lying on the dorsum along the metacarpal bone. The phalanx is disarticulated and the metacarpal head is excised.

Thumb amputation. The thumb represents 50% of the function of the hand. It has the opponens action that assists in grasping. Preservation of any part is the rule. If the metacarpal is all what is left behind, the web between it and the index is deepened by incision to create a new thumb.

Hand amputation. No formal amputations are described since the smallest stump is better than any artificial hand, and the rule is to preserve as much of the hand as possible.

Wrist amputation. Amputation through the wrist permits the most possible pronation and supination and provides better prosthetic control than higher amputation.

Major amputations

Below the elbow amputation. The optimum length is 7 inches from the tip of the olecranon, but even if only a short stump can be achieved, forearm amputation is preferable to above elbow amputation. Equal anteroposterior flaps are fashioned to obtain a terminal transverse scar which will not be drawn up between the two bones.

Disarticulation of the elbow is usually carried out through a single posterior flap or through equal anterior and posterior flaps.

Above the elbow amputation. The optimum length is 8 inches form the acromion process. Even if a very high amputation is necessary, the head of the humerus should be spared since it serves as a support for a prosthesis and maintains shoulder width.

Disarticulation of the shoulder is rarely indicated.

Forequarter amputation is indicated for malignant tumours. This operation entails removal of the whole upper limb with the shoulder girdle.

SURGERY OF THE SPINE AND SPINAL CORD

The structure of the spine and its intimate relationship with the spinal cord and nerve roots are shown in Fig. 57.1.

CHAPTER CONTENTS

Congenital anomalies of the spine

1. A congenitally **wedged vertebra** or hemivertebra may cause a severe degree of scoliosis or kyphoscohosis. Several vertebrae may be fused, especially in the cervical spine.
2. Sacralization of the fifth lumbar vertebra which means fusion of this vertebra with the sacrum. **Lumbarization** of the first segment of the sacrum. Such abnormalities can rarely be incriminated as a cause of backache.
3. **Spondylolisthesis** is a disorder in which there is a defect in the pars interarticularis (articular processes) of the fourth or fifth lumbar vertebrae which allows the vertebral body to slip forwards on the sacrum (Fig. 57.2). **Spondylolisthesis** may be clinically obvious if there is a step in the lumbo-sacral spine. The lumbar vertebrae are more easily felt per abdomen than usual. In early cases of spondylolisthesis physiotherapy, rest and a spinal support are effective. In gross cases of slipping, spinal fusion may be necessary, sometimes with removal of a prolapsed intervertebral disc. Fusion by bone grafting may be achieved by an approach to the affected spines posteriorly, laterally or from the front.
4. Spina bifida.

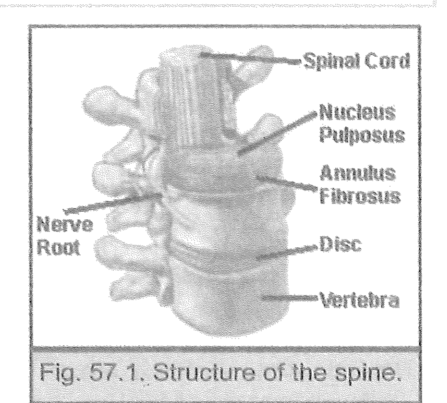


Fig. 57.1. Structure of the spine.


Fig. 57.2. Spondylolisthesis.

Spina bifida

Spina bifida means split or open spine (Fig. 57.3). Folic acid administration during pregnancy may lower its incidence. The most frequently affected area is the lumbar spine.

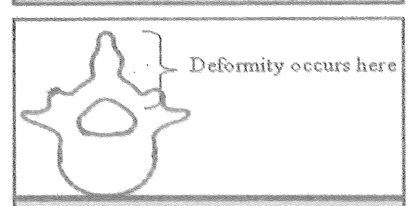


Fig. 57.3. Site of defective vertebral development in spina bifida.

Embryological background

Spinal cord

- The ectoderm forms a neural groove, which then becomes a neural canal.
- The neural tube is separated from the skin to become the spinal cord.

Vertebrae

- The notochord forms the centre of spine, i.e., centre of vertebral body and centre of intervertebral discs (nucleus pulposus)
- The mesoderm around the notochord sends two mesodermal wings which constitute the neural arches which meet in the middle line to form the spinous processes. Thus the vertebra is completely formed and now completely surrounds the cord.

During any stage of development of the spinal cord and vertebral column, arrest may occur, resulting in one type of spins bifida.

Spins bifida may present as an isolated anomaly or in association with other congenital anomalies in the same baby. The common association is referred to by the acronym VACTERL (chapter 37).

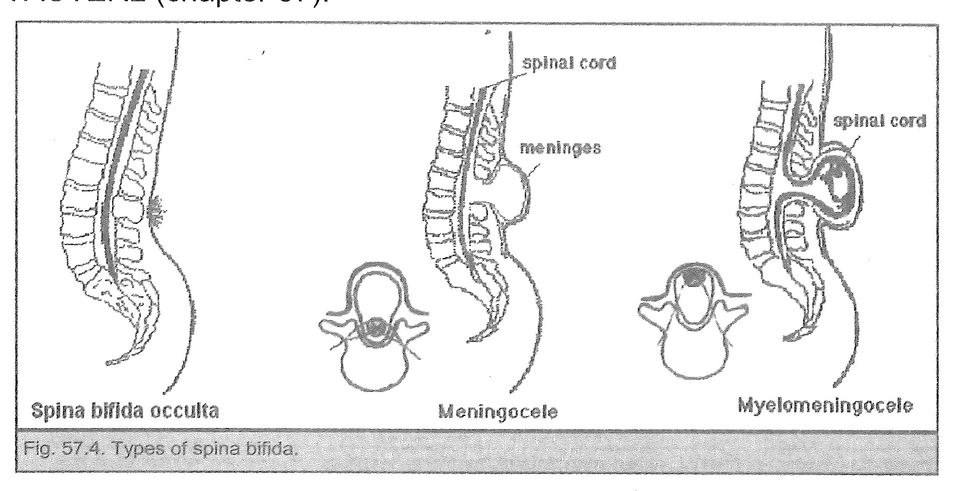


Fig. 57.4. Types of spina bifida.

Types (Fig. 57.4). A bifid spine may be hidden (occult) or manifest.

1. **Spina bifida occulta** (about 10% of population). The development is nearly complete except for a bifid spine. The skin is connected to the meninges by a fibrous band (membrana reuniens). There may be no evidence of the presence of spina bifida or there may be a lipomatous tumour, skin dimple or a tuft of hair. In most cases no problems arise from spins bifida occulta. In a minority of cases, about puberty, incontinence of urine appears (at first nocturnal enuresis), or other nervous manifestations arise. The symptoms arise because traction on the membrana reuniens reaches its maximum at the period of maximum elongation of the vertebral column, i.e., at puberty. Since the meninges are pulled from the back, pressure is exerted on the motor tracts. The tracts responsible for sphincteric control are the last to become myelinated, and will be the first to suffer if traction is applied on the meninges by the fibrous band. Plain X-ray of spine reveals deficient laminae.
2. **Meningocele** is bulging of the meninges only through the spinal defect. It does not contain any nerve tissue.
3. **Meningo-myelocele** (Fig. 57.5) is the commonest type of manifest spina bifida. The normally developed spinal cord or cauda equina lies in the sac and they may

Common association of anomalies VACTERL

- **V** Vertebral
- **A** Anorectal
- **C** Cardiac
- **T** Tracheal
- **E** Esophageal
- **R** Renal
- **L** Limb anomalies

+ Hydrocephalus

The order of frequency of spina bifida types

- **Spina bifida occulta**
  Usually asymptomatic
- **Meningomyelocele**
  Neurological manifestations
  Lumbar swelling
- **Meningocele**
  Lumbar swelling

appear as dark shadows on transillumination. Arnold Chiari malformation is usually present. In this malformation the cerebellar tonsils lie at a level below the foramen magnum and obstruct the flow of CSF from the fourth ventricle, thus producing a concomitant hydrocephalus. If untreated 75% of cases die within the first year of life.

4. **Syringomyelocele** is a rare form where the central canal is dilated.
5. **Myelocele** (complete spina bifida). Here there is failure of fusion of the neural tube. An open spinal plate, which dribbles CSF, occupies the defect. This anomaly is incompatible with life.



Fig. 57.5. Meningomyelocele.

Clinical features and diagnosis

- Antenatal screening
  - Ultrasound.
  - High levels of alpha-fetoprotein in amniolic fluid
- Spina bifida occulta
  - Usually asymptomatic. Accidental finding in X-rays.
  - Lipoma, skin dimple or a tuft of hair over the bifid spine.
  - Rarely urinary incontinence starting at adolescence.
- Meningocoele
  - No neurological manifestations
  - Cystic translucent swelling with an expansible impulse on coughing.
  - The swelling is compressible.
- Meningo-myelocele and syringomyelocele
  - Paraplegia, wasting, or contractures, with sensory loss.
  - Trophic disturbances are marked, particularly perforating ulcers of foot,
  - Other congenital deformities are frequently associated, the commonest and most important is hydrocephalus.

Treatment

- Spina bifida occulta requires no treatment in the majority of cases. If the membrana reuniens produces incontinence, it should be surgically divided.
- In other forms surgery is attempted as early as possible.
  - The operation consists of covering the defect by flaps from the lumbar fascia. Excision is done only in pure meningocele, but the types which contain nerve tissue are not excised.
  - Ventriculo-peritoneal (or atrial) shunt for hydrocephalus.

Fractures and dislocations of the spine

Fractures and dislocations of the vertebrae are important as they may produce spinal cord and nerve root injuries. Fractures are more frequent at the junction of the mobile and rigid parts in the lower cervical and dorso-lumbar regions.

Aetiology

Trauma (Fig. 57.6)

- Hyper-flexion trauma. This is the most frequent type of trauma, e.g., fall of a heavy object on the back of the trunk or the head.
- Hyperflexion and rotation.

- Vertical compression trauma.
  - Fall from a height on the feet (fractures of the calcaneum is a common association) or the buttocks.
  - Fall on the head, e.g., diving in shallow water.
- Hyper-extension injuries are uncommon.

Pathological fractures

- Metastases
- Osteoporosis

Types

According to morphology (Fig. 57.7)

1. **Wedge compression fracture**. Hyper-flexion trauma crushes the vertebral body into the shape of a wedge. The posterior spinal ligaments are intact. The fracture is stable. The cord is undamaged and is in no danger.

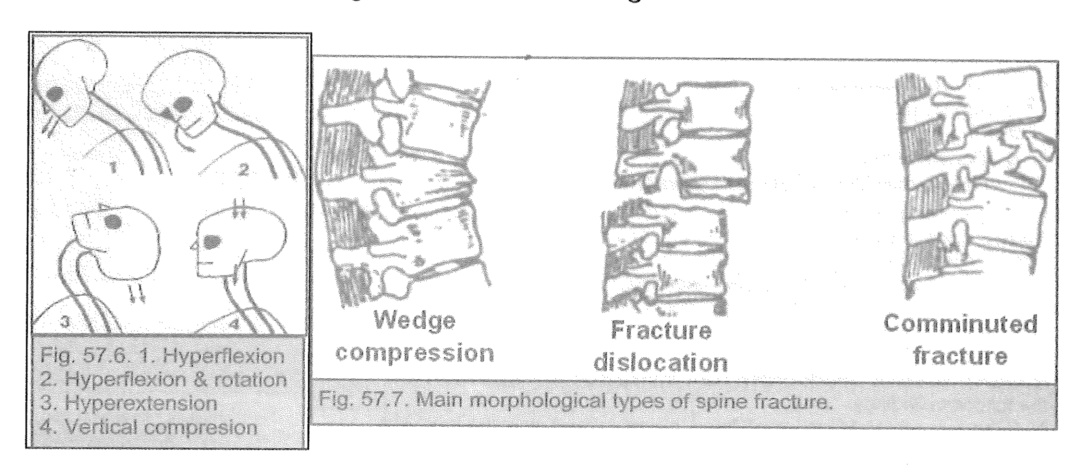


Fig. 57.6. 1. Hyperflexion 2. Hyperflexion & rotation 3. Hyperextension 4. Vertical compresion

Fig. 57.7. Main morphological types of spine fracture.

2. **Fracture dislocation**. If hyper-flexion is accompanied by rotation, one or more of the articular processes breaks. The posterior ligaments are also torn and forward dislocation of a vertebra occurs. Obviously, this is an unstable fracture. Commonly there is damage to the spinal cord and nerve roots.
3. **Dislocation**. Pure dislocation without a fracture is possible in the cervical spine because its articular processes are rather horizontal. The consequences are similar to those of fracture dislocation.
4. **Comminuted (burst) fracture**. This is an uncommon injury. If at the time of a vertical compression the spine is straight, the vertebral body is comminuted. Ligaments are not ruptured and the fracture is stable. Cord or root damage may, however, occur as fragments of the vertebral body are thrust backwards into the spinal canal.
5. Avulsion fractures of transverse and spinous processes are not accompanied by neurological injury and require no special treatment.

Dislocations and fracture dislocations of the atlas and axis vertebrae are especially serious as they may be fatal because of transection of the cord above the level of innervation of the respiratory muscles. Atlas dislocation can occur with or without fracture of odontoid process (hangman's fracture).

According to stability

Stability of spine fractures depends on the integrity of the posterior ligaments; the interspinous, supraspinous and ligamentium flavum.

- Stable fractures have intact posterior ligaments, e.g., wedge compression, comminuted (burst), and avulsion fractures of the transverse and spinous processes.
- Unstable fractures have torn posterior ligaments and are liable to injure the spinal cord and nerve roots. These include fracture dislocations (Fig: 57.8) and pure dislocations.

Neurological injury

Paraplegia or quadriplegia is the result of injury of the spinal cord, nerve roots or both. It is important to diagnose the level of the lesion as well as its nature to know whether recovery is expected or not.

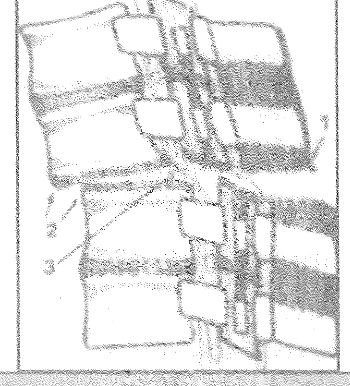


Fig. 57.8. Fracture dislocation is unstable and may injure the cord.
1. Torn posterior ligaments.
2. Displaced vertebra.
3. Crushed spinal cord.

Level of lesion (Fig. 57.9)

The spinal cord ends at the lower border of the first lumbar vertebra.

- Fractures below this level can produce only root lesions of the cauda equina.
- The first sacral cord segment is opposite the lumbo-dorsal junction. Injuries of the spine at the level of the twelfth dorsal or first lumbar vertebra causes paralysis due to combined cord and root damage. It isolates the sacral cord (this is irrecoverable). It will also cause paralysis of the muscles controlling the hip by damage to D12, L1 nerve roots.
- The first lumbar cord segment in adult is at the level of the tenth dorsal vertebra. Injury of the spine at this level isolates the entire lumbar and sacral cord with permanent paralysis of the lower limbs and viscera. Injury of the spine between the tenth and the twelfth dorsal will cause a cord lesion at any level in the lumbosacral cord.
- In cervical spine fractures the segmental level of cord injury corresponds to the level of bone injury. One or two additional nerve roots are also injured. High cervical cord transection is fatal because all respiratory muscles are paralyzed.

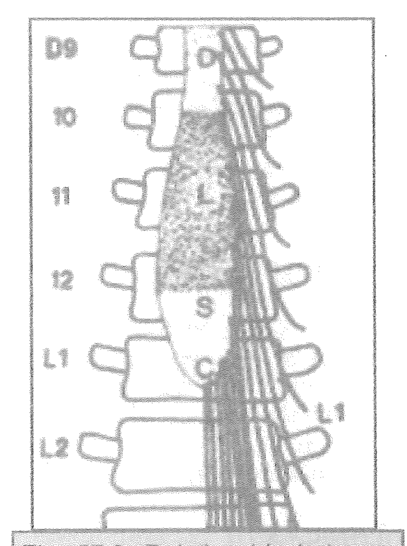


Fig. 57.9. Relationship between vertebral level and spinal cord.

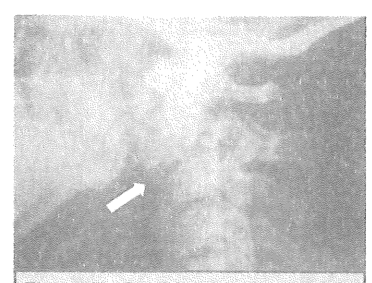
Nature of the lesion

- Spinal cord concussion. Like cerebral concussion and nerve concussion (neurapraxia) recovery is the rule.
- Spinal cord damage by contusion or laceration (transection). These injuries do not recover. The types of spinal cord transaction are:
    - Complete transaction.
    - Hemisection (Brown-Sequard syndrome)
- Cauda equine injury.

Spinal cord transection produces an early stage of spinal shock with complete flaccid paralysis below the line of cord section, loss of tendon reflexes and an atonic distended bladder. Spinal shock lasts for a few days. The cord below the level of transection then recovers reflex function, which results in spastic paralysis and a reflexly emptying bladder.

However, in spinal shock phase cord transaction is suspected with the following findings:

1. If there is complete loss of all forms of sensation below the level of the lesion. In concussion, joint position sense which ascends in the posterior column is preserved.
2. If the sensory loss and paralysis rise after injury indicating ascending edema.
3. A damaged cord, which has not recovered in 48 hours, will never recover.


Fig. 57.10. Dislocation of C2 vertebra. Spinal cord transaction is likely and may cause respiratory paralysis and death.

A very sure sign of cord transection is the appearance of the mass reflex (at 3-6 weeks) and the presence of anal and penile reflexes in the absence of sensation in the legs.

Clinical features

- Spine injury is suspected after any major trauma or if the victim is unconscious after the injury. A cervical collar is applied before moving the victim and the head is immobilized (Fig. 2.4).
- Other injuries of the skull, chest, abdomen, or limbs must be searched for.
- Local signs. The patient is gently rolled onto his side as one piece (chapter 2).
    - A slight kyphosis may be visible or felt.
    - There is local-tenderness over the spinous process. No gap is palpable between the spinous processes in a stable fracture. In an unstable fracture, separation of the spinous processes may be evident.
    - No attempt should be made to test back movements as this may induce paraplegia.
- Neurological assessment
    - Motor power.
    - Sensations.
    - Reflexes.


Fig. 57.11. Wedge compression fracture. In this case the patient has osteoporosis.

Radiography

- Plain X-ray (Fig. 57.11 & 57.12). Besides A-P and lateral views, a special view through the open mouth is needed to diagnose fractures and dislocations of the atlas vertebra. Cervical spine films should show all seven cervical vertebrae and T_1.
- CT scan and MRI must be done to assess encroachment on the spinal cord (Fig. 5712).


Fig. 57.12. MRI showing encroachment on the cord by bone fragment.

Treatment

Initial management

- When fracture of the spine is suspected movements of the spine must be prevented.

- A cervical collar is applied and the head is immobilized during transport and during examination (Fig. 2.4 & 57.13).
- The trauma victim is moved on to the stretcher all in one piece; he should never be lifted by the shoulders and the thighs.

- In the hospital, the patient is nursed on a bed with fracture boards. Shock must be treated. Associated injuries may require urgent treatment (chapter 2).

- The stability of spine fracture depends on the integrity of the posterior ligaments.
- Always suspect spine fractures in victims of major trauma.

Stable fractures

- Wedge compression fracture. The fracture is disregarded. The patient is put to bed and is taught extension exercises for the back muscles. Within a month, he is allowed up. He can resume work in 3 months. A painless, mobile and powerful back is achieved in spite of the fact that the compressed vertebra remains wedge shaped.
- Comminuted fracture. Pain is usually severe and healing is delayed. A plaster jacket is applied in the neutral position (straight spine; not hyperextended) for 3 months. The patient can walk with the plaster on. Spinal exercises must be practiced regularly as soon as the plaster jacket has been applied.
- Avulsion fractures of the transverse or spinous processes. Rest and analgesics.

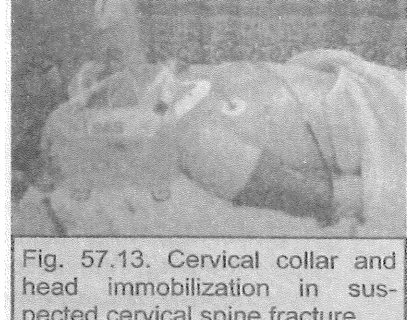


Fig. 57.13. Cervical collar and head immobilization in suspected cervical spine fracture.

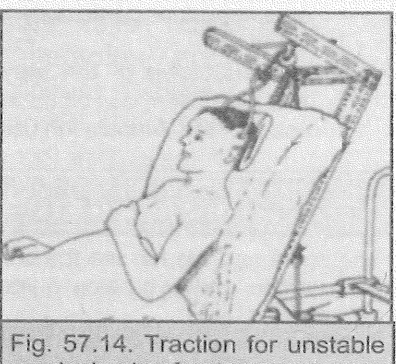


Fig. 57.14. Traction for unstable cervical spine fracture.

Unstable fractures

- **No neurological deficit:** Reduction and immobilization are required to prevent spinal cord injury. This is later followed by physiotherapy for rehabilitation.
  - Cervical spine. Traction using tongs (Fig. 57.14) for 6 weeks is followed by a cervical collar (Fig. 57.15) for one month. Open reduction and internal fixation by plates may be needed.
  - Thoraco-dorsal spine. Reduction by gentle extension is followed by fixation in a plaster cast in mild extension. If closed reduction is not possible, open reduction and internal fixation is the alternative.
- **Evident cord transaction:** Paraplegics are better treated in special centres where the personnel are well trained in their management. The patient is transported with care to avoid further damage to the cord. In most centres a conservative policy is adopted. However, if paralysis is incomplete and particularly if it is increasing or if CT scanning shows fragments of bone encroaching upon the spinal canal, decompression and internal fixation are to be advised. In some centres internal fixation is routinely used for all paraplegics and is said to facilitate nursing and rehabilitation of the patients, but this is controversial.

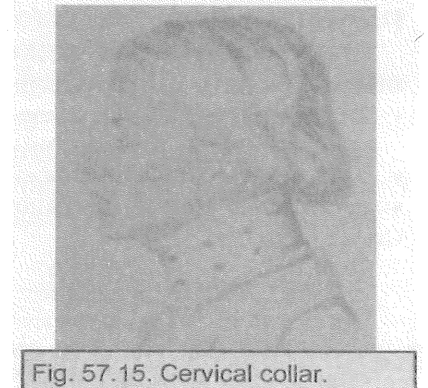


Fig. 57.15. Cervical collar.

Principles of management

- **Postural reduction and** two hourly turning. The position of extension of the spine is maintained by pillows (Fig. 57.16). Plaster casts are contraindicated as they produce pressure sores. Every two hours day and night, the patient is turned as one part on to the other side by trained nurses.
- **Medications**. Corticosteroids are given for the first 48 hours to reduce cord oedema. Prophylaxis against DVT and stress ulceration is advised.
- **Care of the skin**. Bedsores develop rapidly unless the patient is well nursed. The skin is kept clean and dry. The skin is washed, dried and powered. The patient is turned over every two hours. After two or three months the skin becomes tolerant, the fracture has become stabilized, and the patient can turn himself. Established bedsores need general treatment and nutritive diet to build the patient's resistance. Sloughs are removed and daily dressings are applied in preparation for skin grafting.

Fig. 57.16. Spine extension by pillows.

- **Care of the bladder**
  - In the initial stage of spinal shock the paralyzed urinary bladder becomes distended, overflow incontinence eventually occurs. The bladder is kept empty by an indwelling catheter.
  - Later reflex activity of the bladder appears as the detrusor muscle regains tone (automatic bladder) and the bladder empties by contraction of the detrusor muscle and relaxation of the internal sphincter in response to distension. Stroking the side of the thigh initiates bladder contraction.
  - In lesions of the cauda equina, the bladder remains paralyzed and micturition occurs by abdominal straining. The bladder may be emptied by intermittent catheterization every twelve hours, or by an indwelling catheter.
  - Urinary infection is prevented by attention to asepsis during catheterization. Urine is examined bacteriologically and the appropriate antibiotic is given.
  - Patients left with high residual urine need specially investigations including cystography and cystometry. Transurethral resection of the bladder neck or sphincterotomy may be indicated.
- **Bowel training**. An enema is done every third day. Mild laxatives may be needed. The patient is trained to strain and evacuate the bowel.
- **Muscles and joints**. Contractures are prevented by passive movements of the joints. Positioning is also important particularly for equinus deformity. Established contractures may need correction by tenotomy.
- **Rehabilitation of the permanent paraplegic**. The trunk and shoulder muscles are developed so that the patient can sit up and use his arms to turn himself and hold crutches. A wheel chair may be used for life. Ambulation using leg braces and crutches may be possible.

Tuberculosis of the spine (Pott's disease)

The spine and the hip are the commonest two joints to be affected by tuberculosis. The disease is more frequent in children below 5 years of age (75% below 10 years) but may occur at any age. In adults the prognosis is bad. Tuberculosis affects the spine secondarily from a primary focus in the lungs or mediastinal lymph nodes through the blood stream.

Pathology

Site

- The commonest site is the lower thoracic and upper lumbar spine. Usually two (or three) adjacent vertebrae are affected with the discs in between.
- In children the disease starts in the cancellous bone in the centre of the vertebral body.
- In adults the disease starts subperiosteally under the anterior longitudinal ligament.

Complications

1. **Deformity**. In the common type, which affects the vertebral body in a child, TB destroys the inter-vertebral disc and the adjacent surfaces of the vertebral bodies, which slowly collapse and obliterate the intervertebral space. Destruction of the framework of the vertebral bodies results in their collapse and the development of an angular kyphosis called gibbus (Fig. 57.17). Because the destruction is more in the anterior part of the vertebra and disc, away from the spinal cord, the disease leads more to deformity rather than to pressure on the spinal cord and paraplegia, which occurs only in 10% of cases. The deformity is most marked in the thoracic region. In the lumbar and cervical regions which are normally lordotic, there is only obliteration or straightening of that lordosis. The deformity is permanent.

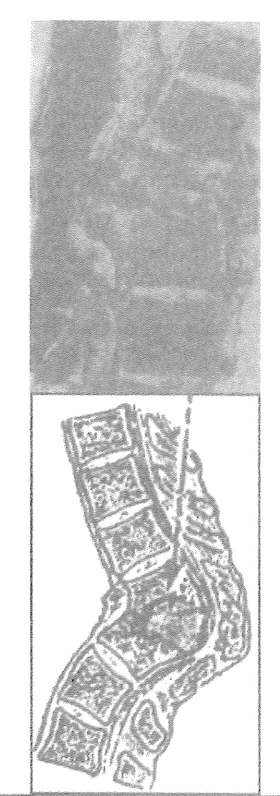


Fig. 57.17. Childhood Pott's disease destroys two adjacent vertebrae and the disc between them.
It causes angular kyphosis.

2. **Cold abscess**. The products resulting from caseation and bone destruction collect in the form of a cold abscess under the anterior longitudinal ligament and on the sides of the spine behind the prevertebral fascia, (paravertebral cold abscess). It spreads along the fascial planes (Fig. 57.18):
    a. In the cervical region, a retropharyngeal abscess bulges in the pharynx in the middle line.
    b. In the thoracic region, it commonly accumulates in the mediastinum but it may pass along the fascial space provided by the intercostal nerve to present on the side of the chest wall or on the sides of the sternum along the perforating branches of the internal mammary artery.
    c. In the lower thoracic and the lumbar regions it passes into the sheath of the psoas muscle to form the famous psoas abscess which presents as a cystic swelling in the posterior abdominal wall, or it may pass under the inguinal ligament to present in the femoral triangle lateral to the femoral vessels with cross fluctuation between the two collections.

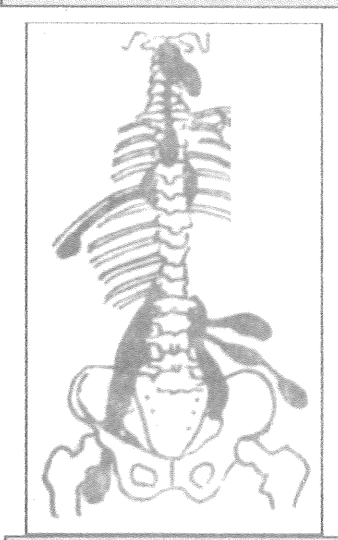


Fig. 57.18. A cold abscess travels along tissue planes to these sites.

3. **Paraplegia** in Potts' disease occurs in 10% of cases. The level of cord affection is usually in the upper dorsal spine, being the narrowest part of the spinal canal, Paraplegia complicating Potts disease is classified into:

a. Early-onset paraplegia which is due to edema, thickening and congestion of the dura or is due to pressure by an abscess or bony sequestrum. It is treated by early anterior decompression and debridement followed by spinal fusion. About 80% recover, usually within a few weeks.
b. Late-onset paraplegia is due to increasing deformity or reactivation of disease or vascular insufficiency of the cord. Investigations should be carried out to establish the precise diagnosis. If MRI shows a block, operative debridement is still worth doing even in late cases. If there is no block, operation is unlikely to be of value.

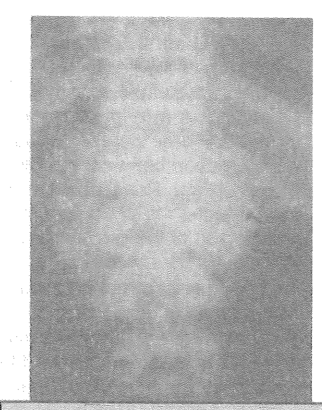


Fig. 57.19. Typical childhood Pott's disease in thoraco-lumbar region destroys two adjacent vertebrae and their disc (narrow disc space. The soft tissue shadow (arrow) is that of a cold abscess.

Clinical features

1. There is marked tuberculous toxaemia; wasting and fever.
2. Local pain which increases on movement. The child walks carefully and slowly to prevent jarring of the spine. Pain may be referred along the nerves to the abdomen.
3. The most important sign (as in all arthritis) is limitation of movements in all directions, thus a child is unable to pick a coin from the ground with the knees straight, he has to bend his knees and on rising he climbs on himself. Rotation and side bending are also limited. In the cervical region the patient supports his head with his hands and rotates as a whole.
4. Kyphosis is angular and is evident if the disease affects the thoracic region. In the cervical and lumbar regions the deformity is masked by the normal lordosis present which is just obliterated and the spine looks straight.
5. A cold abscess should be looked for in the above mentioned sites. A retropharyngeal cold abscess infereres with respiration, swallowing and phonation.

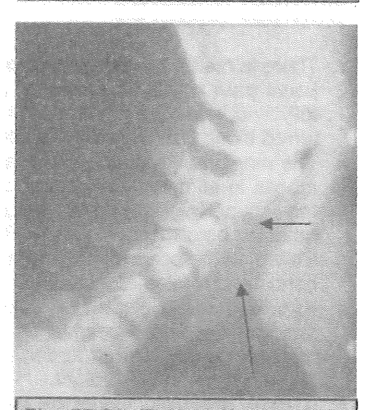


Fig. 57.20. Typical X-ray of cervical spine TB in an adult. The disease affects the anterior subperiosteal part of the vertebra (short arrow) and causes bone destruction without new bone formation. The soft tissue shadow anterior to spine is caused by a cold abscess.

Radiography

- **Plain X-ray** (Fig. 57.19 & 57.20)
  - Two or more vertebrae are affected.
  - Intervertebral disc space is narrowed and is later completely lost.
  - Adjacent surfaces of the vertebrae are irregular.
  - Vertebral bodies may be collapsed with areas of bone destruction.
  - No bone sclerosis or new bone formation.
  - Paravertebral abscess shadow is usually present.
- CT scan

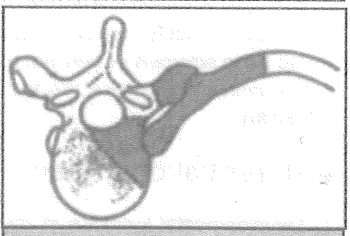


Fig. 57.21. The excised area for antero-lateral decompression.

Differential diagnosis

1. Developmental kyphosis in children. Potts kyphosis is characterized by being angular.
2. Other causes of back pain in adults.

Treatment

Treatment is essentially conservative

Conservative treatment

1. Anti-tuberculous medications for 9 months.
2. Rest.
3. Good nutrition.
4. Spine support by plaster cast for a short period. When the disease become quiescent this is changed into a spine brace.
5. Periodic clinical, haematologic and radiographic assessment. Under conservative treatment most cases are cured. Early paraplegia patients respond favourably to this treatment in 60% of cases. Late Potts paraplegia is due to increasing severity of kyphosis and responds neither to conservative nor surgical treatment.

Surgery

Indications

Early Pott's paraplegia with:

1. No sign of recovery after 3-4 weeks of conservative treatment.
2. Paraplegia getting worse in spite of conservative treatment.

Aim of surgery

Decompression of the spinal cord

Technique

The caseous material and dead bone are removed by either of two methods:

1. **Costo-transversectomy:** In this operation the posterior ends of one or two ribs and the corresponding transverse processes of the vertebrae are excised and the cold abscess is evacuated.
2. **Antero-lateral decompression**: In this operation, in addition to costo-transversectomy the pedicles and part of the vertebral bodies are excised to achieve decompression (Fig. 57.21).

Later treatment

1. Life-long follow-up.
2. Spine fusion may be needed after clearance of infection, using bone graft.

Intervertebral disc prolapse

The intervertebral disc is made up of central nucleus of a jelly like substance (nucleus pulposus) enclosed under tension by a fibrous ring (the annulous fibrosis), the whole being enclosed between fibrocartilagenous plates above and below. The intervertebral discs act, as shock absorbers and allow normal mobility between the adjacent vertebrae (Fig. *57.1)*.

Aetiology

1. Sudden strain with the spine in an unguarded position (80%) will rupture the annulus leading to protrusion of the nucleus either in the midline or laterally.
2. Degeneration of the annulus (20%).

Level

Disc protrusions occur in the most mobile segments of the spine.

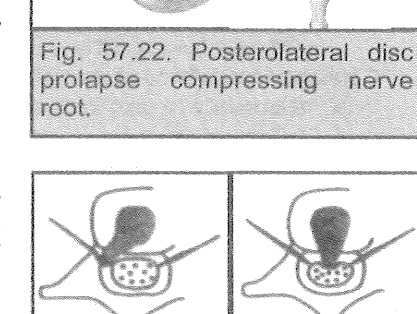
1. 80% occur in the lumbar region, particularly *L415* and L5/S1.
2. 19% occur in the cervical region particularly *C516* and C6/7.

Prolapse direction (Fig. 57.22 & 57.23)

1. Posterolateral leading to compression on nerve roots.
2. Posteriorly leading to compression on the spinal cord.
3. Into the vertebral body (Schmorl's node) is rare.

Osteoarthritis of intervertebral joints may follow degeneration and prolapse of the disc.

Fig. 57.22. Posterolateral disc prolapse compressing nerve root.



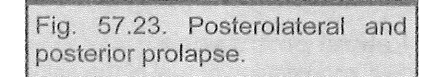


Fig. 57.23. Posterolateral and posterior prolapse.

Lumbar disc prolapse

Clinical features

1. Low back pain. Typically, while lifting a heavy object or stooping the patient is seized in back pain. The spinal muscles go into painful spasm and the patient describes himself as locked in agony. He becomes unable to straighten up.
2. Root pain due to compression of nerve roots. This pain radiates down the back of the lower limb and is increased by coughing.
3. Motor and sensory changes will occur according to the affected nerve root.
   a. $L_{4/5}$ disc will affect the 5th lumbar root. Pain and sensory loss may affect back of the thigh, lateral aspect of the leg and dorsum of the foot. There may weakness of ankle dorsiflexion.
   b. L_5/S_1 disc will affect the first sacral root. Pain or sensory loss will affect the back of the leg and sole of the foot. There may be weak planter flexion of the ankle and absent ankle jerk.
4. The patient adopts a scoliotic position and has a limping gait to reduce pressure on the nerve root (Fig. 57.24).
5. Straight leg raising or flexion of the spine increases the pain due to compression of the nerves against the protrusion.

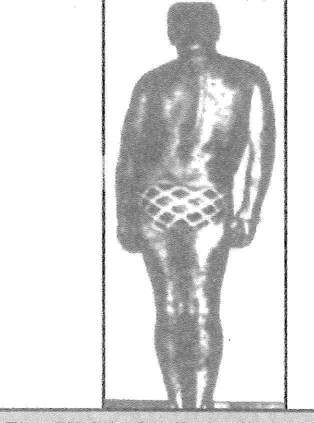


Fig. 57.24. Scoliotic tilt.

Investigations

1. **Plain X-ray of spine** (PA and lateral views).
   a. To exclude bone diseases that cause similar manifestations.
   b. Later it will reveal narrowing of the disc space. In chronic cases there may be osteophytes at the margins of the vertebrae.
2. **CT scan and MRI** can accurately localize the site of disc prolapse or spinal cord compression.

3. Myelography was previously used for the diagnosis but has been replaced by CT or MRI.

Treatment

Conservative treatment

- Rest in a bed on boards placed under the mattress to give a rigid support for a period of 2-4 weeks relieves the pain in the majority of cases.
- Analgesics, usually an anti-inflammatory agent.
- Physiotherapy after recovery from the acute attack.
- Weight reduction.
- Avoidance of sudden flexion strains on the spine.

Surgical treatment

Indications

- Persistent symptoms.
- Recurrent attacks of severe pain.
- Evidence of motor or sensory affection or sphincteric disturbances.

Aim

Removal of the prolapsed nucleus pulposus.

Approach to the prolapsed disc is either by

- Laminectomy. Through a vertical midline incision in the back. The lamina of corresponding vertebra (or vertebrae) is removed. This gives access to the dura which is retracted medially to expose and remove the prolapsed material.
- Microdiscectomy. Here no bone is removed but access is gained by incising the tissues between adjacent laminae. The operation can be conducted by endoscopic surgery.

Causes of recurrence of pain after surgery for a lumbar disc:

- Removal of a disc from a wrong level.
- Presence of another undiagnosed disc prolapse.
- Presence of interstitial neuritis of the nerve roots due to prolonged compression.
- Osteoarthritis of the intervertebral joints, in this case spinal fusion should be performed during the operation for removal of the disc.

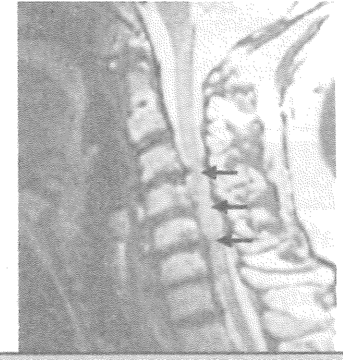


Fig. 57.25. MRI showing cord compression by prolapsed discs at 3 levels (arrows).

Cervical disc prolapse (Fig. 57.25)

Lateral protrusions

1. Compression of half of the spinal cord leads to Brown-Sequard syndrome.
2. Compression on nerve roots depends upon the affected disc level:
   a. $C_{5/6}$ disc compresses the 6th cervical root. Pain and sensory loss affect the outer border of the arm and dorsum of forearm. There may be weakness of the biceps and diminished biceps and supinator jerks.
   b. $C_{6/7}$ disc compresses the 7^{th} cervical root. Pain and sensory loss affect the back of upper arm and forearm. There may be weakness of the extensors of the fingers and diminished triceps jerk.

Movements of the spine aggravate the pain while rest or traction of the spine alleviates it.

Midline protrusions

Compression of the anterior part of the cord may lead to quadriplegia. Compression of the anterior spinal artery may affect the pyramidal and spinothalamic tracts.

Treatment

1. A collar made of plastic on felt may be worn to limit movements of the neck.
2. More severe cases with nerve root compression require traction by the physiotherapist for about 20 minutes every day.
3. In very severe cases, traction of the neck is maintained for as long as the patient can stand it by a head halter.
4. Analgesics.
5. If there is evidence of cord compression with developing quadriplegia and urinary incontinence, urgent decompression by laminectomy may be needed, followed by spinal fusion.

Spondylosis

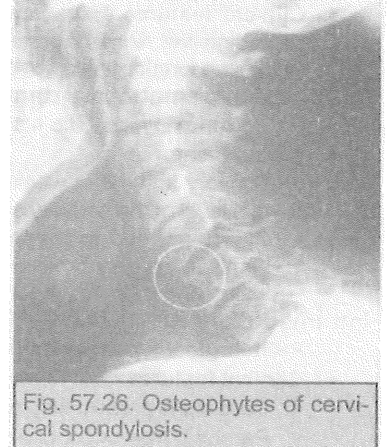
Pathology

Spondylosis of the spine is a degenerative condition.
It is often a part of generalized osteoarthritis. More localized spondylosis may follow trauma, though the actual incident may have occurred many years previously and have been forgotten. The cervical and lumbar vertebrae are particularly affected.

Clinical features

- Pain, usually in a nerve-root distribution
- Limitation of movement
- Stiffness.

Many of the symptoms and signs of spondylosis are the same as those presenting in prolapse of intervertebral disc. An element of disc prolapse is a common feature of spondylosis, though usually radiographic and clinical features indicate a more widespread affection of the vertebrae than those found in disc prolapse alone.


Fig. 57.26. Osteophytes of cervical spondylosis.

Radiography

Plain X-ray reveals osteophytic lipping of the edges of the vertebrae, especially obvious in the lateral view (Fig. 57.26). Osteophytes are particularly liable to form around the vertebral foramina through which nerve roots pass. Disc degeneration manifests itself as narrowing of the space between adjacent vertebrae.

Treatment

The treatment for spondylosis is much the same as for prolapsed intervertebral disc.

- **Analgesics** as anti-inflammatory agents are given to relieve pain.
- In mild cases physiotherapy and exercises to build up the strength of spinal extensor muscles are recommended. Traction for varying periods may be valuable.
- **Spine support**

  - o Lumbar spine. Abdominal corset with steel stays inserted posteriorly to limit movements of the lumbar spine.
  - o Cervical spine. Cervical collar.
  - o In some cases pain may be so severe as to require immobilization in a plaster jacket.
- **Surgery**. Operation is needed for the severest persistent symptoms, or for evidence of advancing nerve pressure. Laminectomy for decompression of neural elements is followed by spine fusion with bone grafts.

Spinal tumours

As in the brain, any space occupying lesion around the soft encaged spinal cord will produce the same symptoms, signs and even radiological appearance of a tumour.

Fig. 57.27. CT showing metastasis to a vertebra.

Site

Spinal tumours may be extradural or intradural. Intradural tumours may be intramedullary (that is inside the cord) or extramedullary.

Extradural tumours

- **Pathological types**
  - o The commonest tumours are secondary deposits (Fig. 57.27), usually from breast, lung or prostate cancer.
  - o Sarcoma, chordoma and neurofibroma are also found. A neurofibroma may be "dumb-bell" in shape, the smaller part lies within the neural canal while the larger part projects through an intervertebral foramen and encroaches on the thorax.
- **Clinical features**. Extradural tumours present with symptoms and signs of irritation of nerve roots preceding the signs of pressure on the cord. Other conditions, such as a prolapsed intravertebral disc in the lumbar or less commonly the cervical region, may produce identical symptoms.



Fig. 57.28. MRI showing an intramedullary tumour.

Intradural extramedullary tumours

- **Pathological types**. The most important are meningiomas and neurinomas.
- **Clinical features**
  - o Sensory and motor weakness. The anterior or posterior roots tend to be involved first; the proximal level of the sensory and motor changes depends on the site of the tumour.
  - o Symptoms due to pressure on the cord itself occur later, there may be the Brown-Sequard hemisection phenomenon, with weakness and loss of vibration sense on the same side as the lesion and loss of pain on the opposite side. Paraplegia will follow if pressure is not relieved.

Intramedullary tumours (Fig. 57.28)

- **Pathological types**. Intramedullary tumours are usually gliomas or ependymomas.
- **Clinical features**

- Cord signs present first, while root pains tend to occur later.
- Paralysis may occur on both sides, or sometimes may be of the crossed type, where there is anaesthesia on one side and paralysis and hyperaesthesia on the other.
- Dissociated sensory loss may occur.
- Urinary incontinence usually appears early.

Investigations

1. Plain X-ray of the spine, anteroposterior, lateral and oblique views should be taken. Erosion of bone or some other bony abnormality ia seen in nearly 50% of patients with spinal tumours.
2. CT scan and MRI.
3. Myelography has been largely superceded by CT scan and MRI.

Treatment

Laminectomy is performed and, if possible, the tumour is removed. Removal of the tumour and laminectomy help in decompression of the cord. Acute emergency operation.

Most frequent site	
Spondylolisthesis	**L_5/S_1**
Spina bifida	**Lumbar**
Fracture	**Dorsolumbar and cervical**
Pott's disease	**Dorsolumbar**
Disc prolapse	**Lumbar ($L_{4/5}$ & L_5/S_1) and cervical ($C_{5/6}$, $C_{6/7}$)**

MINOR PROCEDURES

CHAPTER CONTENTS

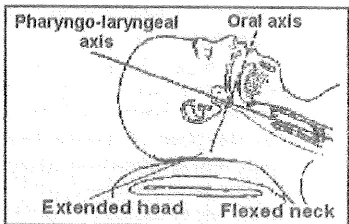
Tracheal intubation

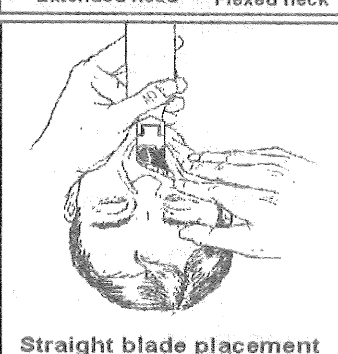
Indications

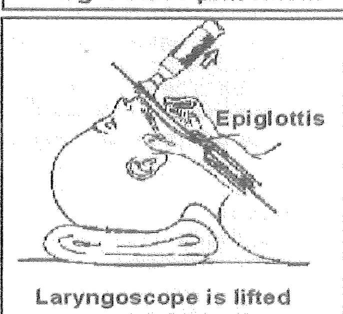
1. For administration of general anaesthesia.
2. To allow for mechanical ventilation in cases failure.

Technique (Fig. 58.1)

1. Frequently an induction agent, e.g. thiopental, and a neuromuscular blocking agent, e.g., succinylcholine are administered to facilitate intubation.
2. Position: Supine with flexion of the neck and extension of the head. For trauma victims fracture of the cervical spine should be excluded before moving the neck in this position because an unstable fracture can cause spinal cord injury.
3. Pre-oxygenation with mask ventilation.
4. Gentle downward pressure on the thyroid cartilage by an assistant facilitates intubation.
5. Standing at the head of the patient, the laryngoscope handle is grasped in the left hand and the blade is gently placed in the right side of the mouth.
6. The tongue is moved to one side of the oral cavity while advancing the blade toward the larynx.
7. The entire laryngoscope is lifted straight anteriorly to expose the vocal cords.
8. With the cuff deflated, the tube is passed under vision into the right side of the mouth and through the vocal cords. A 6 or 8 mm tube is usually suitable for adults.
9. The endotracheal tube is placed so that the cuff is just distal to the cords. The balloon is inflated with 5-10 ml of air.
10. Correct positioning is checked. The chest is inspected for symmetrical movements with insufflation of air, and breath sounds are auscultated.







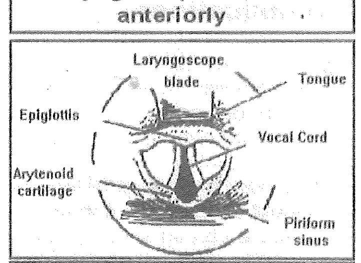


Fig. 58.1. Steps of tracheal intubation.

Complications

1. Minor lacerations to the tongue, lips, and gums.
2. Dental damage.
3. Oesophageal intubation.

Central venous catheterization

Indications

1. Central venous pressure (CVP) monitoring.
2. Parenteral nutrition.
3. Long-term infusion of drugs, e.g., cytotoxic agents.

Contraindications

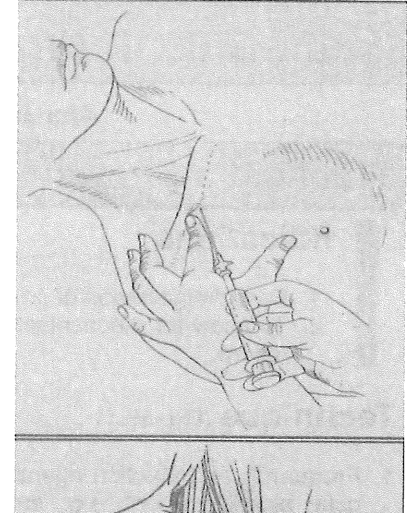
1. Coagulopathy.
2. Burns or skin infection at the site of entrance.

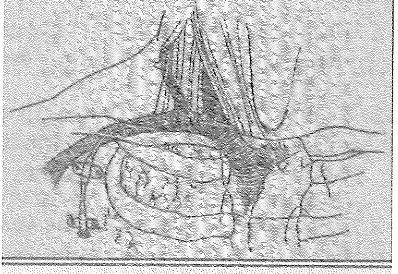
Central venous catheterization is usually performed through the subclavian or internal jugular vein.

Technique of subclavian vein catheterization

(Fig. 58.2)

1. Position is supine with head down tilt.
2. Sterile preparation of subclavian area, using an antiseptic and drapes. The operator uses mask, gown and gloves.
3. Local infiltration of xylocaine at an area 0.5 cm below the clavicle and 2 cm lateral to its intersection with the first rib. *Always aspirate before injection.*
4. A wide (18 gauge) needle is attached to a syringe and is used to puncture the skin in the anaesthetized area. While aspirating, the needle is advanced slowly horizontally (parallel to the floor) in the direction of the suprasternal notch. Entery into the subclavian vein is signaled by free aspiration of blood.
5. The syringe is removed while keeping a finger over the needle to avoid air embolism. A guide wire ("J" wire) is passed through the needle with its tip directed towards the heart. The needle is kept in place until the wire passes into the vein (Seldinger technique). The wire must pass without resistance.
6. The needle is then removed and the central venous catheter is introduced over the guide wire to the length of 15cm on the right and 18cm on the left.
7. The wire is removed, the catheter flange is sutures to the skin, and the area is covered by a sterile dressing.
8. A chest x-ray confirms placement into superior vena cava and rules out pneumothorax.





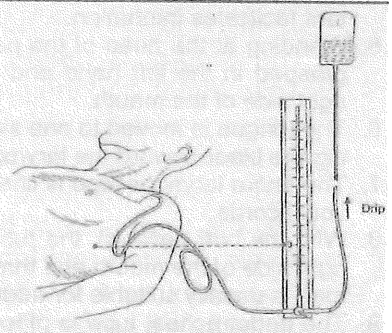


Fig. 58.2. Steps of subclavian vein catheterization

Technique of internal jugular vein catheterization (Fig. 59.3)

1. Position is supine with head down tilt. The patient's head is turned 45° contralaterally.
2. The area to be anaesthetized is at the middle of posterior border of sternomastoid muscle which is grasped firmly and is elevated slightly to fix the internal jugular vein.
3. While maintaining aspiration the needle is passed slowly deep to the muscle in a direction between its sternal and clavicular heads.
4. Other details of the technique are the same as for subclavian vein catheterization.

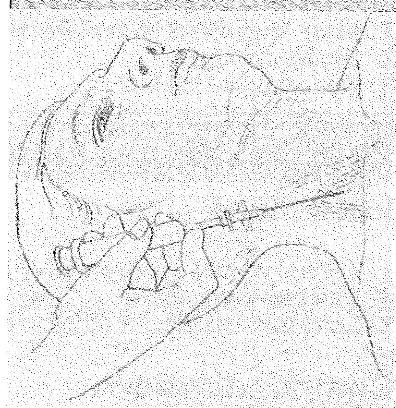


Fig. 58.3. Internal jugular vein catheterization

Complications

1. Arterial puncture.
2. Air embolism may occur either during insertion or later in the course of therapy.
3. Pneumothorax caused by pleural injury.
4. Malpositioning.
5. Infection of the catheter causes septicaemia. The catheter should be removed and its tip is sent for culture and sensitivity. Until then broad-spectrum antibiotics are started.

Venous cut-down

Indications

Venous cut down is done when percutaneous access to the venous system cannot be gained.

For infants the long saphenous is the preferred vein, while for adults any accessible vein, e.g., cephalic, antecubital, or the long saphenous vein can be used.

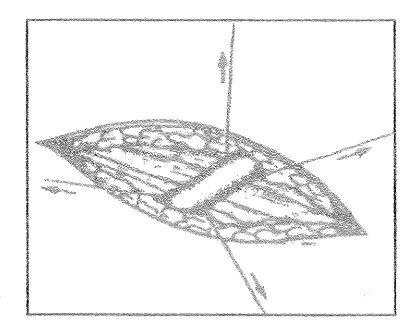
Technique of long saphenous cut down

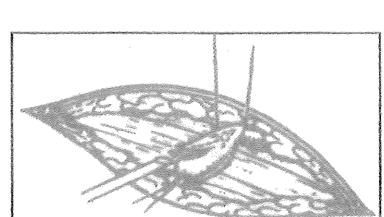
In adults the long saphenous vein is consistently located 1 cm anterior and 1 cm superior to the medial malleolus (Fig. 58.4).

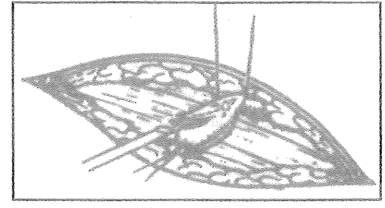
1. Skin preparation and draping of the ankle region.
2. Infiltration of the skin over the vein with xylocaine.
3. A full-thickness transverse 2 cm incision is made through the anaesthetized skin,
4. A curved artery forceps with a fine tip is used to dissect the vein. The saphenous nerve should be separated from the vein to avoid its injury.
5. A ligature is passed around the proximal end of the dissected vein and another around its distal end.
6. The vein is ligated distally, leaving the suture in place for traction.
7. A small transverse venotomy is made with fine-tipped scissors and the IV cannula is passed into the vein.
8. The proximal ligature is tied around the vein, being careful not to occlude the cannula.
9. Intravenous infusion is started.
10. The wound is closed with interrupted sutures. antibiotics ointment and then a sterile dressing are applied.

Complications

1. Bleeding.
2. Infection and thrombophlebitis.
3. Injury of the saphenous nerve.







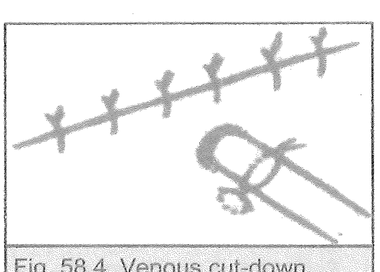


Fig. 58.4. Venous cut-down.

Urethral catheterization

Indications

1. Urine retention.
2. Urine output monitoring for a critical patient.
3. Irrigation of blood clots, e.g., after prostatectomy.
4. To allow healing after open urinary bladder surgery or after minor extraperitoneal ruptures.
5. Intravesical chemotherapy for superficial bladder cancer.
6. As a stent after urethral surgery.
7. For cystourethrography.

Contraindications

Suspected urethral injury. Suspicion is raised by either:

1. Blood at urethral meatus.
2. Perineal ecchymosis.

Technique

For men (Fig. 59.5)

1. Sterile gloves are used. A disinfectant solution is applied to the external genitalia. Sterile draping.
2. Injection of 10 ml of sterile xylocaine gel through the meatus facilitates the procedure and reduces patient's discomfort.
3. The penis is held with the left hand, and is placed on a stretch perpendicular to the body to straighten the anterior urethra.
4. A well lubricated Foley catheter (size 16 F is recommended for an adult) is held in the right hand, introduced through the meatus and is advanced gently and steadily up the urethra until urine is returned.
5. The balloon is inflated with 10 ml saline.
6. The catheter is connected to a urine collection bag.
7. An enlarged prostate narrows and kinks the urethra, thus making the procedure difficult. A Coude-tip catheter is often helpful to negotiate the angle between the bulbous and membranous urethra.

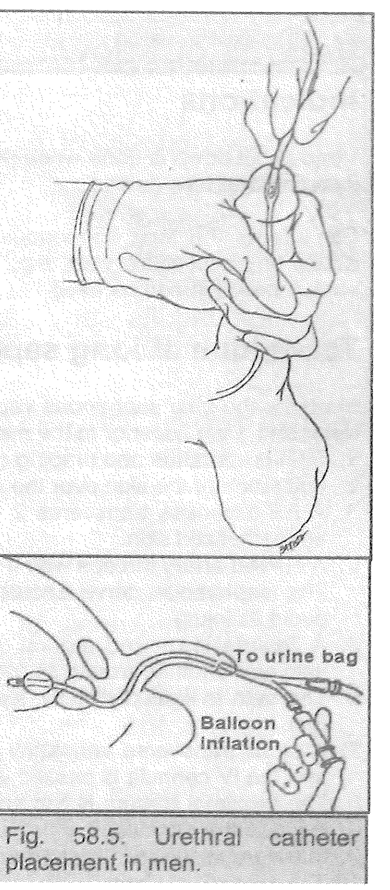


Fig. 58.5. Urethral catheter placement in men.

For women

1. Supine position with the hips flexed and abducted, and the knees flexed.
2. The left hand spreads apart the labia minora to expose the urethra meatus and the right hand passes the catheter up to the bladder.
3. There is no need to pass the catheter up to its end. It usually brings urine after a short passage.

Complications

1. Urethral injury particularly in cases with prostate enlargement. A false passage is evidenced by the appearance of blood at the meatus. Attempts at catheterization are abandoned.
2. Excessive diuresis after the relief of acute retention may produce hypovolaemia.
3. Urinary tract infection.

Nasogastric tube (NGT)

Indications

1. Aspiration of upper GI contents in patients with acute gastric dilatation, paralytic ileus, small bowel obstruction, gastric outlet obstruction or acute pancreatitis.
2. Upper gastrointestinal bleeding.
3. Enteral feeding.

Technique

1. Position. Sitting or supine. Ask patient to flex the neck.
2. The tube is lubricated, is introduced into a patent naris, and is advanced posteriorly into the nasopharynx.
3. The patient is asked to swallow. With swallowing the tube is advanced to the oesophagus and then into the stomach.
4. Placement in the stomach is confirmed by aspiration of gastric contents, or by auscultation of injected air.
5. The tube is carefully taped to the nose in a way not to press on the naris.
6. The tube is connected to a collection bag. Low intermittent suction is applied as well. Patency is ensured by injecting 20 ml saline/4 hours.

Complications

These are usually minor but then get commoner with prolonged use of the tube.

1. Ulceration and necrosis of the naris.
2. Oesophageal reflux, oesophagitis, oesophageal erosion and even oesophageal stricture.
3. Mouth breathing, mouth dryness, and possibly parotitis.
4. Interference with ventilation and coughing, retention of bronchial secretions, atelectasis, and pneumonia.
5. Loss of fluids.
6. Other complications include traumatic epistaxis, sinusitis, and otitis media.

PRE AND POSTOPERATIVE MANAGEMENT OF THE SURGICAL PATIENT

Preoperative assessment and preparation

CHAPTER CONTENT

Assessment of the surgical patient aims at minimizing the operative risk and ensuring a safe conduct of any surgical procedure.

The process comprises a careful history intake, meticulous clinical evaluation and ordering the necessary investigations.

The following needs to be assessed:

1. Cardiovascular system:

- Enquiry and assessment for hypertension, ischaemic heart disease, arrythmias, heart failure and medications should be performed.
- ECG, stress ECG or echocardiography may be performed.
- All the previously mentioned problems should be corrected pre-operatively after consultation of the cardiologist. Elective surgery should be delayed for 6 months after an attack of myocardial infarction. Patients with valvular disease or prosthetic valves should receive pre-operative antibiotics to guard against subacute bacterial endocarditis.

2. Respiratory system:

- Enquire about history of smoking, chronic bronchitis, bronchial asthma, and obstructive airway disease.
- Chest X-ray and pulmonary function tests may be needed.
- Smoking should be stopped for one month before elective surgery. Chest physiotherapy and expectorants are advised.

3. **Neurological system:** Enquire about epilepsy, cerebrovascular accidents and transient ischaemic attacks.

4. History of drug intake:

- Antiplatelets and anticoagulants: aspirin should be stopped for ten days before elective surgery to avoid troublesome bleeding. The decision should be taken in cooperation with the cardiologist. Non-steroidal anti-inflammatory drugs should be stopped two to three days before operation. Oral anticoagulants should be stopped for 3-5 days before surgery. If anticoagulation is necessary prescribe low molecular weight heparin.
- Steroids; the dose should be carefully increased shortly before operation to tide the patient over the stress of surgery. The dose can be tapered postoperatively to the previous preoperative levels.
- Contraceptive pills should be stopped 4 weeks before elective surgery to minimize the risk of thromboembolism.
- Insulin, antibiotics, antihypertensives, digitalis or other drugs should be clearly recorded.
- History of drug allergy, e.g. penicillin.
- History of drug abuse; intravenous abusers are liable to harbour hepatitis B or C viruses as well as HIV.

5. **History of previous operations** and/or complications of anaesthesia e.g. jaundice after halothane anaesthesia or prolonged apnea after operation due to succinylcholine apnea.
6. **Assessment of the nutritional status:** Remember that both undernourished and morbidly obese patients withstand surgery poorly and are liable to post-operative complications (Chapter 9).
7. **Patients with preexisting liver disease** as liver cirrhosis, should be classified according to the Child-Pugh classification. The operative risks and mortality increase steadily with the increase of the Child grade. The three main risks to such patients include:
   - Bleeding: it is better to optimize the prothrombin time, and concentration and INR before surgery. Vitamin K and fresh frozen plasma may be necessary.
   - Hepatocellular failure: every effort should be exerted to avoid massive bleeding, hepatotoxic drugs, severe dehydration or sepsis which may contribute to tip the balanced patient into decompensation.
   - Sepsis: any septic focus and/or infection of the ascitic fluid (spontaneous bacterial peritonitis) should be treated before surgery.

8. **Management of diabetic patients.**

Surgery and anaesthesia increase the circulating catecholamines, ACTH and growth hormone, all of them antagonize the action of insulin. The situation is more severe in diabetics with previous insulin deficiency resulting in much more pronounced hyperglycaemia than in normal patients. Without insulin, lipolysis is stimulated and ketone bodies may increase resulting in metabolic ketoacidosis.

General rules for the management of diabetic patients:

- They should be considered as high risk patients even with minor procedures.
- Meticulous survey of the patient is essential to detect any possible organ affection by diabetes e.g. checking all accessible pulses.
- Minimize the duration of fasting to avoid hypoglycaemia.
- The patient should be kept on just the hyperglycaemic side.
- Diabetic patients controlled by insulin may require larger doses of insulin to tide them over the stress of surgery (Table 59.1).

Table (59.1): Simplified scheme for pre and postoperative management of diabetic patients

Type of the patients diabetes	Minor surgery	Major surgery
Controlled by diet	No specific precautions	Measure blood glucose 4 hourly. If >220 mg/dl, give glucose-insulin infusion
Controlled by oral drugs	Omit the drugs on the morning of operation then restart when oral feeding is allowed postoperatively	Omit oral drugs and monitor blood glucose 2-hourly, if >220 mg/dl, start glucose-insulin infusion
Controlled by insulin	Omit the morning dose of insulin and start glucose-insulin infusion	Same

A standard glucose infusion regimen in two separate simultaneous infusion lines:

Glucose infusion	Regular insulin in saline infusion
1 litre of glucose 5%, 100 ml/hour	50 units of insulin in 500 ml saline, 1 unit/10 ml/hour

The above basal rate of glucose-insulin infusion can be changed according to the blood glucose level to keep the blood glucose level on the hyperglycaemic side. Potassium is added to the glucose infusion as required.

Checklist before transfer to the operating theatre:
- Patient identity including full name and hospital number outlined in bracelets.
- Marking of the side of operation, e.g., inguinal hernia.
- Informed consent.
- Check that the patient is fasting at least six hours before operation.
- Prohylactic antibiotics and anticoagulants are administered if indicated.

Post-operative care

Postoperative care of the surgical patient has two phases:
1. Immediate postoperative care (the recovery phase), in the recovery room.
2. Care in the ward until discharge from hospital.

Phase (I): The recovery phase:

Immediately after surgery patients require close monitoring in a recovery area, adjacent to the operating room. The following items should be clearly fulfilled before discharge of the patient to the ward:
1. The patient should be awake and alert.
2. The patient should be able to maintain the airway.
3. Stable vital signs.
4. The ability to call for help as needed.
5. No obvious surgical complication as bleeding from the wound or via the drain.
6. Pain control.
7. Nausea/vomiting controlled.
8. Normothermic, avoid hypothermia as it leads to impaired clotting and cardiac arrhythmias.

In the recovery room continuous ECG monitoring and pulse oximetry are performed. The pulse oximeter displays the oxygen saturation (normally 95-100%) which reflects the oxygenation of the patient. Oxygen is given by a mask or by nasal catheters.

Phase (II): Care in the ward:

For each patient keep records of the following:
1. Fluid balance charts; input and output charts.
2. Medication sheet; revise and update:
    a. Antibiotics.
    b. Analgesics.
    c. I.V. fluids.
    d. Anti-coagulants.
    e. Anti-diabetic drugs.
    f. Anti-hypertensives.
3. Investigations as required.

Review the following aspects at bedside
1. General comfort of the patient; psychological support and ambulation.
2. Pain control:

- Inadequate pain control leads to increased sympathetic tone and liability to myocardial infarction. It also leads to impaired respiratory muscle functions and impaired cough reflex.
- Methods of pain control:
    - Narcotic drugs.
    - Non-steroidal anti-inflammatory drugs.
    - Epidural analgesia.
    - Patient controlled analgesia.

3. Vital signs: check whether there is tachycardia, hypotension, orthostatic hypotension, or fever.
4. Respiration; check absence of dyspnea or tachyponea; judicious chest exercise.
5. The surgical wound: check for edema, redness, discharge, or dehiscence.
6. Urine output: normally 0.5-1 ml/Kg/hour, check the urethral catheter.
7. Drains: check drain output, kinking, blocking, slippage, leaking fluids from the drain site or bleeding.
8. Blood sugar control: check the sliding scale of insulin.
9. Nutritional status: daily weighing, check oral intake.
10. Check the pressure areas at the sacrum or heels for edema or bed sores.

Postoperative complications:

Surgical complications, although part of the surgical practice, should be avoided largely by careful preoperative assessment and by meticulous surgical technique. Post-operative complications can be divided into:

- **General** complications.
- **Local** complications in the wound.
- Complications **specific** to the performed operation.

General complications:

- Cardiac complications: include hypertension, arrythmias, myocardial infarction, and heart failure.
- Respiratory complications: (Chapter 28).
- Deep vein thrombosis and pulmonary embolism (Chapter 14).
- Nausea and vomiting.
- Thrombophlebitis of a peripheral on central venous line.
- Retention of urine and urinary tract infection.
- Fever; the following are the causes of postoperative fever:
    - Reaction to surgery may occur in the first 24 hours.
    - Pulmonary complications are the usual cause in the first few days.
    - Surgical site infection usually presents on the 4^{th} or 5^{th} day, but it may be delayed.
    - Thrombophlebitis of a peripheral or central venous line.
    - Deep vein thrombosis and pulmonary embolism.
    - Urinary tract infection.

- After abdominal surgery remember leakage of an anastomosis or development of an intra-abdominal abscess.
- Neurological complications as stroke or transient ischaemic attacks.

Local complications in the wound

- Seroma.
- Haematoma.
- Wound dehiscence.
- Wound infection.
- Hypertrophic scar and keloid.

Complications specific to the performed operation

Each surgical procedure has its specific complications which the surgeon tries to avoid by meticulous surgical technique. The following are the complications which may follow abdominal surgery.

- Acute gastric dilatation.
- Paralytic ileus.
- Adhesive intestinal obstruction.
- Leakage of an intestinal or gastric anastomosis. This may lead to peritonitis, intra-abdominal abscess or faecal fistula.
- Intra-abdominal abscess (pelvic, interloop, or subphrenic).
- All the previous complications will lead to nausea and vomiting.

PRINCIPELS OF MODERN SURGICAL PRACTICE

CHAPTER CONTENTS

Contemporary medical practice requires a doctor who has certain capabilities. Acquiring enough knowledge and technical or clinical skills is not enough for the graduate of medical schools to be considered as an efficient and reliable doctor. In the following chapter some of the principles which govern modern medical practice will be briefly discussed; the following items will be mentioned:

- Medical ethics
- Communication skills
- Evidence based medicine
- Clinical trials and methods of research
- Audit

Medical ethics

"Ethics is a code of values which guides our choices and actions and determines the purpose and the course of our lives'.

- Ethics is an important component of the international standards of medical education.
- Graduates should be able to behave ethically at all times towards patients and their relat*ives in a* manner consistent with the ideals of the profession and to use ethical principles in clinical decision making and research.

The two main ethical concerns in medical practice:

(A)Patients rights:

1. Autonomy versus paternalism.
2. Informed consent.
3. Beneficence. The doctor should have the obligation to maximize benefits to the patients.
4. Non-malbeneficence. The doctor should have the obligation not to do harm to the patients.
5. The right to refuse treatment.
6. Advanced medical directives.

(B)Patient-doctor relationship:

1. Truth telling.
2. Confidentiality.
3. Justice and resources allocation.

(A) Patients rights:

1. Autonomy versus paternalism

- Autonomy means self control. It means the patient's right to participate in decisions about the diagnosis and treatment.
- The doctor-patient interaction relationship is now more of a therapeutic partnership in which information is collected, options discussed and opinions shared.
- At the end of this process the patient makes a choice as to how his condition should be managed.

- In certain clinical situations autonomy can be replaced by Paternalism. Paternalism means to overrule in individual's decision on the ground that he is not capable of making the correct decision for himself; children, unconscious patients, demented patients, mentally retarded patients, emergency situations where delay in decision making may affect the patients life.

2. **Informed consent:** Surgeons have a legal as well as a moral obligation to obtain consent for treatment based on appropriate levels of informations to the patients.
   - The patient has to sign an agreement and accept the consequences and responsibilities.
   - Patients have the responsibility to communicate openly and to comply with the agreed upon treatment program.
   - Written consent infront of a witness offers some defense against a claim that consent was not given.
   - For every procedure, the patient should be offered an explanation of the problem and possible solutions, and then their consent asked.
   - A description of the recommended treatment or procedure.
   - A description of the alternative, including other treatments or procedures, together with the risks and benefits of these alternatives.
   - The likely results of no treatment.
   - The probability of success, and what the physician means by success.
   - The time period during which the patient will not be able to resume his or her own activities.
   - A process rather than a signature on a form.
   - Informed consent is needed in the following activities:
     a. Medical or surgical treatment.
     b. Medical research.

The components of informed consent include:

(a) Voluntariness (without any pressure) this is of particular relevance in the area of Medical Research.
(b) Competence (the patient can make decisions for himself).
(c) Disclosure the patient has to know everything concerning his disease and the plan of management, cost effectiveness of alternative choices. Exploration should be in simple language the patient can understand, with no scientific terms.
(d) Consent (the patient has to sign an agreement accepting the consequences).
(e) The concept of consent to treatment is the core of patient rights and is fundamental to the doctor/patient relationship.
(f) Touching a patient without his/her consent is legally an assault.

Waiving the consent:

- In some situations medical care can be provided to adult patients incapable of consenting provided the action is in their best interests (incompetent patients) e.g. prisoners, demented people, comatosed patients.
- In these cases, the consent regarding medical care can be signed either by the family or any other authorized person after discussing the matter with a competent specialized doctor in front of witness.

Informed consent for research:

"Consent of ethical committee should be available"

Ethics for Clinical Trials:

- Experiments should be based upon prior animal studies.
- The experiment must be for the good of society.
- The results cannot be obtained by other means.
- Informed consent.
- Subject can terminate her/his involvement.
- Equitable selection of participants.
- Competent investigators.
- Processes to monitor safety of intervention.
- The risks versus the benefits.

3. **Beneficence** (the obligation to maximize benefits to the patients):
   - The principle requires that actions and intentions are in the best interest of the patient.
   - Competent patients should be allowed to decide for themselves how to balance harms and goods.

4. **Non-malbeneficence** (the obligation not to do harm to the patients):

   Examples of harms to the patients:

   A. **Misdiagnosis:**
      a. If the doctor did not take an adequate history, did improper clinical examination and have not seen the patients records.
      b. The doctor did not perform the necessary investigations.
      c. Doctors have a responsibility to keep up to date, to maximize the benefits and minimize the harm for their patients.

   B. **Medical negligence:** Some surgical reasons for allegations of negligence:
      a. Operating on the wrong side, e.g. hernia. Good recording and preoperative marking of the correct side are essential.
      b. Amputation of the wrong digit or limb.
      c. Leaving swabs or instruments in the patient. Swab count and on-table X-rays are relevant.
      d. Removing the wrong organ in paired organs as the kidneys.
      e. Operating on the wrong patient when patient identification is inadequate. Accurate recording of the identity and wearing bracelets for identification are essential before induction of anaesthesia.
      f. Transfusion of the wrong blood bag i.e. of other patients.

5. **The right to refuse treatment:**
   - Competent adults have a right to refuse medical treatment, even if this refusal results in death or permanent injury.
   - If a patient is refusing beneficial treatment, the doctor needs to make two judgments before accepting that the patient has the right to refuse:
      a. Judgment 1: i.e. is the patient competent?
      b. Judgment 2: has anyone influenced the patient to such an extent that the patient's decision is not totally free?

6. **Advanced directives:**

   Some patients requests should be respected e.g. do not resuscitate, do not give me blood transfusion, I am ready to donate my organs after death.

(B) Patient-doctor relationship

1. **Truth telling:** In general terms, it is correct to give your patients sufficient information to make an informed choice. The patient has the right to know the nature of his disease, suggested plans for management and the advantages of each including cost effectiveness.

2. **Commitment of confidentiality:** There are three possible reasons for respecting medial confidences:
    a. Because of the private nature of much of what a patient discusses with the doctor. Patients might not seek medical help if they did not believe that doctors would respect their confidences.
    b. Autonomy and respect for privacy. People should have the right in controlling who knows what about them. Thus, it is the right of the patient to ask the doctor to keep some information confidential.
    c. The character of the doctor. Patients often assess the actions of their doctor in the light of a character ideal.

Conflict between confidentiality and the interests of others:

There are situations in which others may be harmed if the doctor does not breach confidentiality. For example:

- A patient with uncontrolled epilepsy may not inform the driving license authority and may continue to drive.
- A patient may wish to keep the fact that he is HIV positive secret from his wife.

The rule is that the doctor's duty is to strictly maintain confidentiality except in specified circumstances:

a. Information is shared with other doctors, nurses or health professionals participating in caring for the patient.
b. Information regarding the patient's health may sometimes be given in confidence to a close relative e.g. in cases of suicidal tendency.
c. information may be disclosed to comply with a statutory requirement, for example notification of an infectious disease, child abuse, suicidal tendency, aggression to the public.
d. information may be disclosed where it is so ordered by a court.

3. Justice and Resources Allocation

The allocation of resources on the basis of clinically irrelevant factors such as religion, age or gender orientation is prohibited.

We ought to identify those interventions, which offer the best value for money (cost-effectiveness) and divert resources to them.

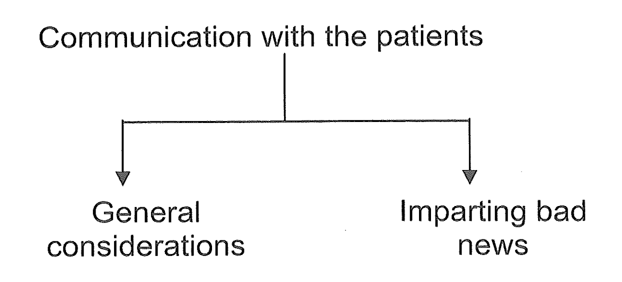
Ethical Professional Attitudes which should be fulfilled:

- Honesty, integrity and trustworthiness.
- Critical self-appraisal (including recognition of limitations and errors).
- Sympathy and compassion.
- Respect for the dignity of patients as people.
- Respect for the roles of other healthcare professionals in the care of the patient.
- Responsibilities of the medical professional towards the local and global community.
- Provide care to patients who are unable to pay and advocate access to health care for members of under served populations.
- Commitment to clinical competence and lifelong education.

Communication skills

An efficient doctor should have the talent of good communication with the patients and their relatives, his colleagues and with the nursing staff.

Communication skills



Communication with colleagues

- Surgical colleagues
- Anesthetists
- Nursing staff
- Administrators
- Radiology and laboratory colleagues

Communication with the patients

- **General considerations:**
    - Surgical patients are usually apprehensive and anxious.
    - Patients expect the doctors to behave professionally and cordially.
    - The pleasing and sympathetic tone of the doctor is important.
    - Avoid casual dresses, and unethical words.
    - When discussing important information with the patients:
        - It should take place in a quiet atmosphere and surroundings.
        - The presence of another colleague and the attending nurse is helpful.
        - Use clear words appreciated by the patient when explaining a surgical situation.
        - Write down the results of the meeting with the patients and/or relatives.
- **When interviewing patients:**
    - Use open questions as often as possible particularly at the beginning fo interview.
    - Avoid leading questions or asking several questions at once.
    - Learn to rephrase a question using simpler language.
    - Careful listening is important.
    - Respect the patients sensitivity, modesty, privacy and culture.
    - An intelligent attitude is required in dealing with depressed, anxious or aggressive patients.
- **When imparting bad news:**
    - Before giving bad news, consider to whom it should be given, who should give it, when it should be given and what are the likely consequences of giving it.
    - Let the relatives be gradually informed about the progression of the case.
    - Use familiar and clear words to explain the situation to the relatives.
    - Allows the relatives to ask questions and discuss the progress of the cause.
    - Never conceal errors or complications.
    - Both sympathetic and professional attitudes are important.

Communication with colleagues:

It should follow the spirit of a team work. All communications with such colleagues should be clearly written and recorded preferably in an electronic file.

On studying the relative contribution of the history (good communication skills), physical examination and investigations to the final diagnosis (100%); it was concluded that:

- Diagnosis made on history alone in 82% of cases.
- Diagnosis changed after physical examination in 9% of cases.
- Diagnosis changed after investigations in 9% of cases.

The above data stress the prime importance of communication skills in achieving a correct diagnosis.

Evidence based surgery

As a simple definition, evidence based surgery is the process of systematically finding, appraising and using recent research findings as the basis for clinical decisions.

The practice of evidence based surgery aims at integrating the personal experience on one hand and the best available and scientifically approved evidence on the other hand. Such scientifically approved evidence is extracted from the body of information which has been evaluated and scrutinized to extract a scientifically valid evidence. The randomized controlled trials (RCTs) constitute the best source of 'evidence'. The best well conducted trials which have been critically evaluated according to certain parameters in a process known as "critical appraisal" are the sources of the best clinical evidence (level I evidence).

Critical appraisal ensures that the design of the trial avoids or minimizes the factors which may affect the truth or validity of the actual outcomes; chance, bias, confounding: the presence of external factors that affect the results.

The strength of evidence is graded in three levels:

- **Level 1 evidence**: deduced from well conducted randomized clinical trials or the result of meta-analysis of several trials.
- **Level 2 evidence:** deduced from several trials, but with some weak points in randomization and/or the design of the trials.
- **Level 3 evidence**: this is not extracted from randomized trials but from circumstantial or unproven evidence.

Accordingly, when we decide to follow such evidence, internationally recognized recommendations, A, B and C in decreasing order of power are agreed.

Grading of recommendations:

- **Recommendation A:** derived from level 1 evidence and should be followed.
- **Recommendation B:** derived from level 2 (◀ j↕ p.. evidence and may or may not be followed according to the patients benefits.
- **Recommendation C:** derived from level 3 evidence. It may be followed only when resources are not adequate to follow a better recommendation.

Clinical **guidelines** represent part of the practice of evidence based surgery. They are simply agreed evidence based protocols of management of clinical situations. Advantages of guidelines:

- They provide an evidence based answer to a specific clinical question
- They aid in decision making.
- They standardize aspects of care with reduction in medical practice variations.
- They help make the most cost effective use of limited resources.
- They facilitate education of patients and health care professionals.

Disadvantages or concerns about guidelines:

1. The evidence used may be weak or irrelevant.
2. They may not be applicable to every patient or clinical situation.
3. Clinicians may feel that they are deprived of the autonomy of decision making
4. There may be geographic or demographic limitations to the applicability of guidelines in different situations.

Evidence based surgery in actual practice requires the following:

1. The spirit of team work with regular meetings and peer reviews
2. Powerful computer
3. Medical secretary
4. Powerful internet connections with access to the important web sites concerned with evidence based practice. Experience is required to perform systematic reviews of the literature extracting the evidence from well conducted clinical trials or meta-analysis of several trials.

Research design and methodology

A good research depends on careful planning, proper implementation and good interpretation of the findings.

Research problem:

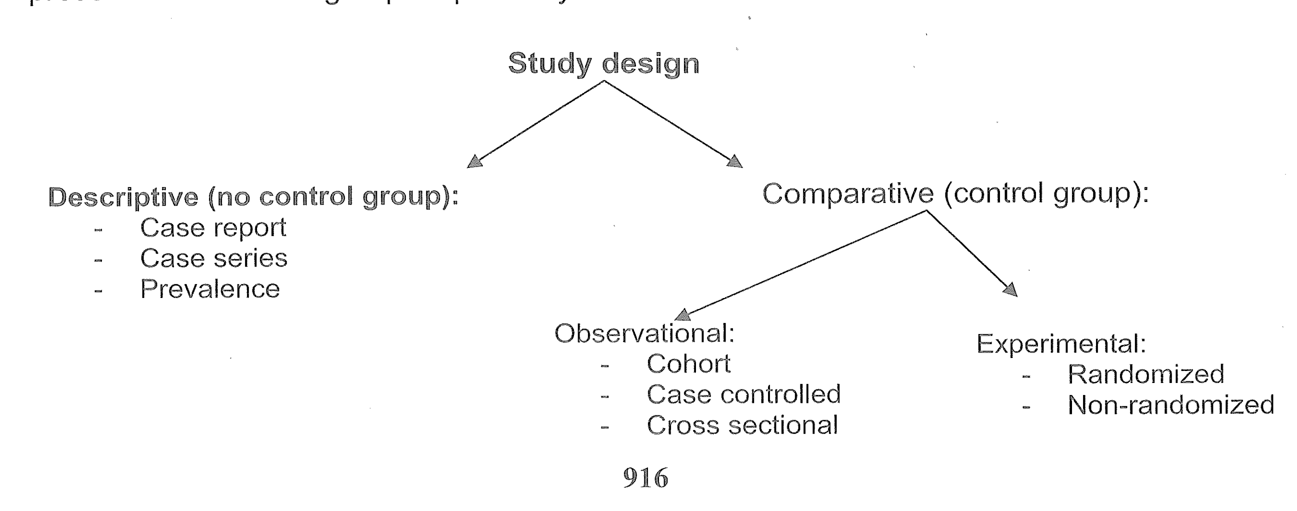
The first question we need to answer is what is the objective of performing a certain research. A good research topic should fulfill certain criteria:

1. It deals with a prevalent problem, so that the results will have a useful impacts.
2. It should not have been previously performed.
3. The study should be accepted by the scientific committee.
4. The results should be applicable.
5. Ethical acceptability: study individuals must consent to participate in the research and they should benefit from the study. Study individuals should have all their needed medical care and they need to be excluded from the research if it interferes with their medical care.

Depending on the research question, a suitable study design should be selected.

Types of research designs:

Researches can be either descriptive or analytical depending on the absence or presence of a control group respectively.



Descriptive studies (no control group)

- Case reports: description of an uncommon observation.
- Case series: description of a series of observations.
- Prevalence studies: describes the prevalence of a certain disease or phenomenon.

Analytical or comparative studies (control group):

They are subdivided into observational and experimental studies:

1. Observational studies:

The investigators report passively their observations without assigning exposure or intervention. Observational studies are further divided into:

a. **Cohort study**: prospective, (measuring an outcome by following up forwardly exposed and non-exposed people).
b. **Case control study**: retrospective, (starting with outcome and looking back in the past searching for an exposure or a risk factor).
c. **Cross-sectional studies** (a snap shot in time): studying all variables without follow up or looking in the past.

Cohort study:

In clinical research, a cohort is a group of subjects followed prospectively over time.

To study the effect of smoking on the probability of developing lung cancer in a prospective cohort study: the investigators choose or define a sample of subjects (cohort). They then subdivide them according to the exposure studied into exposed and unexposed groups (smoking and non-smoking groups). They then measure other characteristics in each subject that might predict the development of the outcome, follow all the subjects up periodically to detect the occurrence (or non-occurrence) of the outcome of interest.

The excess risk of the outcome due to a certain exposure in cohort studies is estimated by a parameter called the relative risk (RR).

Case control study:

Case control studies start from the end (i.e. after developing the outcome of interest). Thus, a group who already developed the outcome of interest (e.g. lung cancer) and a group without the outcome are chosen. Then, we study the prevalence of previous exposure to risk factors (smoking) through interviews or medical records if available. If the prevalence of exposure is higher among cases than among controls, then the exposure is associated with increased risk of the outcome.

Cross-sectional studies:

In a cross sectional design all measurements are made at once with no follow up in time (snap shot in time). Steps include drawing a sample from the population, then looking at the distribution of variables within that sample, trying to associated variables.

2. Experimental design:

An experimental design is a design in which there are:

- More than one group.
- Investigators are the ones who assign the intervention to the groups. i.e. the investigators decide which group to start the experimental intervention and which to start the control intervention. When the groups are allocated using a random scheme the study is called a "randomized" controlled study or trial, while when the groups are allocated based on any non-random scheme i.e. selection it is called a non-randomized controlled study or trial.

The randomized controlled trial (RCT):

This clinical trial is a prospective study comparing the effect and value of intervention against a control inhuman being.

The following are important factors that must be considered and followed by investigators running randomized controlled clinical trials in order to prove the effectiveness of therapeutic interventions like new drugs or new surgical procedure.

1. Randomization:

Randomization is a process by which every subject in the enrolled study population has an equal chance to be allocated to either the intervention or control group. It excludes selection bias that result from directing certain patients to a group and other patients to the other group based on any "non-random" scheme.

Methods of randomization:

- Coin toss
- Sealed envelopes
- Random number tables

2. Allocation concealment:

It is concealing (hiding) the allocation sequence from those assigning the groups until the end of assignment. It prevents researchers from directing certain participants to a given group (selection bias). Inadequate concealment exaggerates the treatment effect by 30-40%.

3. Measuring the outcome:

The comparison groups should be followed up equally throughout the course of the study. In each of the groups, the response variable is measured and recorded for all subjects in the same manner and frequency.

4. Blindness:

Blindness is ensuring that a person remains unaware of the type of intervention a subject has been allocated to till the end of the study. It is particularly valuable in studies involving subjective outcomes, e.g. pain relief, mood elevation, or nausea. It is important because it leads to decrease in the tendency to report more favorable outcomes in the intervention group and less favorable outcomes in the control group. In addition blinding of investigators is important in order to decreases bias in measuring disease outcomes.

Trials are often described as:

1. Single-blind: Blinding the subjects participating in the trial.
2. Double-blind: Blinding the subjects and investigators (clinicians, interviewers, laboratory personnel).
3. Triple-blind: It involves blinding the subjects, investigators and committee responsible for monitoring outcome as well as persons who perform data entry, analysis and statistics.

Presenting and publishing an article (an overview)

- The key to both presentation and publication is to decide the ***MESSAGE*** you want to present in a clear form.
- In a power point presentation limit the number of lines in each slide, highlight the most important point in each slide and use animations, pictures or graphs to stress and pinpoint the message.

- Follow accurately the instructions to the authors when you submit a work for presentation in a national or international journal.
- Convention dictates that the articles are submitted for publication in IMRAD (Introduction, Materials and Methods, Results and Discussion) form:
  - **I- Introduction:** A brief background of the study should be presented with the aims of the work outlined.
  - **M- Methods:** The methodology and study design should be given in detail. It is important to avoid bias. New procedures should be detailed in full.
  - **R- Results:** They are best shown diagrammatically using tables and figures if possible.
  - **D- Discussion:** It is important not to repeat the introduction or repeat the results in this section. The study should be interpreted intelligently. The authors should state whether the results fit into or alter the views of other published research. They may suggest further procedures to reach a conclusion.

Audit

The term audit represents the critical appraisal of the care given by clinicians. It has been defined as "the systematic, critical analysis of the quality of medical care including the procedures used for diagnosis and treatment, the use of resources and the resulting outcome and quality of life for the patient."

Three main aspects of audit are important:

1. **Structure**: the quantity and type of available resources.
2. **Process**: defines what is done to the patient e.g.:
   a. Adequate pre-operative assessment.
   b. The way an operation was performed.
   c. Adequate documentation.
   d. Compliance with the consensus policies of the hospital and/or international practice with the aim of achieving the best quality of practice.
3. **Outcome**: the "result" of the clinical intervention, either success or failure should be documented e.g.:
   a. Morbidity and mortality meetings (a conclusion should be reached as regards the cause and its avoidance).
   b. Patient satisfaction.

Hints on audit:

- Adequate and accurate medical recording is important. It should be in a way agreed by the treating physicians and administration.
- It is an organized team approach.
- It is well-known that errors do occur in medical practice. The auditing process aims at minimizing the errors, analyzing the causes and the avoidance of future occurrence. A level of high quality medical care is the target.
- Another aspect of audit is to compare the outcomes of one institute with other centers "comparative audit". It is not uncommon to modify the practice to comply with the agreed standards of best practice.
- The auditing process and peer reviews maintain obvious educational advantages at all levels
- Auditing is important in managing adverse events or occasional undesired results aiming at improving the quality of practice.